EXHIBIT A

LUCAS COUNTY COMMON PLEAS COURT
CASE DESIGNATION

TO: Bernie Quilter, Clerk of Courts

CASE NO. _____

JUDGE G-4801-CI-0201804752-000
Judge
MICHAEL R GOULDING

The following type of case is being filed:

- ☐ **Professional Malpractice**
  - ☐ Legal Malpractice (L)
  - ☐ Medical Malpractice (M)
- ☐ **Product Liability** (B)
- ☐ **Other Tort** (C)
- ☐ **Workers' Compensation**
  - ☐ State Funded (D)
  - ☐ Self Insured (K)
- ☐ **Administrative Appeal** (F)
- ☐ **Commercial Docket**

By submitting the complaint, with the signature of the Attorney, the Attorney affirms that the name of person with settlement authority and his/her direct phone number will be provided upon request to a party or counsel in this matter

Other Civil

- ☐ Consumer Fraud (N)
- ☐ Forfeiture
- ☐ Appropriation (P)
- ☐ Court Ordered
- ☑ Other Civil (H)
- ☐ Certificate of Title
- ☐ Copyright Infringement (W)

This case was previously dismissed pursuant to CIVIL RULE 41 and is to be assigned to Judge _____, the original Judge at the time of dismissal. The previously filed case number was CI_____.

This case is a civil forfeiture case with a criminal case currently pending. The pending case number is_____, assigned to Judge _____

This case is a Declaratory Judgment case with a personal injury or related case currently pending. The pending case number is_____, assigned to Judge _____

This case is to be reviewed for consolidation in accordance with Local Rule 5.02 as a companion or related case. This designation sheet will be sent by the Clerk of Courts to the newly assigned Judge for review with the Judge who has the companion or related case with the lowest case number. The Judge who would receive the consolidated case may accept or deny consolidation of the case. Both Judges will sign this designation sheet to indicate the action taken. If the Judge with the lowest case number agrees to accept, the reassignment of the case by the Administration Judge shall be processed. If there is a disagreement between the Judges regarding consolidation, the matter may be referred to the Administrative Judge.

Related/companion case number_____ Assigned Judge_____

_____ _____
Approve/Deny Date Approve/Deny Date

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IN THE COMMON PLEAS COURT OF LUCAS COUNTY, OHIO
GENERAL DIVISION

THE STATE OF OHIO, ex rel.
MICHAEL DEWINE
OHIO ATTORNEY GENERAL
Environmental Enforcement Section
30 East Broad Street, 25th Floor
Columbus, Ohio 43215,

Plaintiff,

v.

3M COMPANY
c/o Corporation Service Company
50 West Broad Street, Suite 1330
Columbus, OH 43215;

TYCO FIRE PRODUCTS LP
c/o CT Corporation System
4400 Easton Commons Way, Suite 125
Columbus, OH 43219;

CHEMGUARD, INC.
c/o The Prentice-Hall Corporation
System, Inc.
251 Little Falls Drive
Wilmington, DE 19801;

Civil Action No. **G-4801-CI-0201804752-000**
Judge
MICHAEL R GOULDING

COMPLAINT WITH JURY DEMAND

BUCKEYE FIRE EQUIPMENT COMPANY
A Haon Corporate Agent, Inc.
29225 Chagrin Blvd., Suite 350
Pepper Pike, OH 44122-4633;

NATIONAL FOAM, INC.
c/o The Corporation Trust Company
1209 North Orange Street
Wilmington, DE 19801; and

ANGUS FIRE ARMOUR CORPORATION
c/o The Prentice-Hall Corporation System, Inc.
251 Little Falls Drive
Wilmington, DE 19801,

Defendants.

TABLE CONTENTS

I. INTRODUCTION

1. The State of Ohio, by its Attorney General Michael DeWine ("Plaintiff" or "Ohio" or the "State"), brings this action against Defendants 3M Company f/k/a Minnesota Mining and Manufacturing Company ("3M"), Tyco Fire Products LP ("Tyco"), Chemguard, Inc. ("Chemguard"), Buckeye Fire Equipment Company ("Buckeye"), National Foam, Inc. ("National Foam"), and Angus Fire Armour Corporation ("Angus Fire"), collectively, the "Defendants," for damages to Ohio, including compensatory and punitive damages, recoverable at law or in equity, and for declaratory and injunctive relief, to remedy Defendants' violations of law.

2. This case centers on the widespread environmental contamination and pollution of Ohio's natural resources caused by Defendants' products, with poly- and per-fluoroalkyl substances ("PFAS"), chief among them perfluorooctanoic acid ("PFOA") and perfluorooctane sulfonic acid ("PFOS").

3. PFOA and PFOS are highly fluorinated organic acids, each with eight carbon atoms.

4. PFOS and PFOA have unique characteristics that render their widespread existence "one of the most seminal public health challenges for the next decades," as recently proclaimed by a senior official with the U.S. Center for Disease Control.

5. These chemicals are mobile and persistent: once introduced into the natural environment, they travel long distances and degrade very slowly over long periods of time, if at all. They bioaccumulate and biomagnify: they are absorbed by and build up in living organisms, such as marine animals and ultimately humans. And these chemicals are highly toxic: exposure to PFOA and PFOS is associated with many adverse health effects, including cancer, high cholesterol, thyroid disease, adverse reproductive effects, and preeclampsia.

6. Each of the Defendants designed, manufactured, marketed and sold aqueous film forming foam ("AFFF") products that contain or break down into toxic PFOS and/or PFOA when the AFFF products are used as intended, resulting in significant environmental contamination and pollution.

7. AFFF is capable of extinguishing fires involving fuel or other flammable liquids that cannot be effectively extinguished with water alone. To suppress such fires, AFFF is mixed with water and aerated to form a foam solution that is sprayed onto the fire. Accordingly, if used as intended and as designed by Defendants, Defendants' AFFF products – and the toxic chemicals they contain – are released directly into the environment, seeping into groundwater and soil, and traveling long distances to cause further, widespread environmental contamination.

8. On information and belief, AFFF is the only PFOA/PFOS application in wide usage that, when used as intended, releases these toxic chemicals directly

into the environment to cause further, widespread and harmful environmental contamination.

9. Over the past nearly five decades, AFFF products have been most heavily used not to fight active fires, but for many thousands of firefighting training exercises on military installations and air bases, at civilian airports, and at local firefighting facilities. During each such firefighting or training event, thousands of gallons of AFFF foam solution laced with toxic PFOS and/or PFOA may be used, introducing these chemicals into the natural environment as a result, including in Ohio.

10. The Defendants knew or, at a minimum, should have known about the environmental dangers the ordinary and intended use of their AFFF products posed. Indeed, by the late 1970s, 3M had confirmed internally that PFOS and PFOA had been detected in human blood, in other words, that it had spread far beyond the immediate site of their applications, and were "more toxic than anticipated."[1]

11. The company, however, withheld information concerning these chemicals' toxicity from the U.S. Environmental Protection Agency ("EPA") and

[1] **Exhibit ("Ex.") 1**, "Organic Fluorine Compounds in Blood Chronology" dated October 19, 1977, produced in the Minnesota PFAS Litigation at 3M_MN00000479; **Ex. 2**, 3M Central Analytical Laboratory Report dated August 4, 1978, produced in the Minnesota PFAS Litigation at 3M_MN02343995; **Ex. 3**, 3M Toxicity Study Report dated November 10, 1978; **Ex. 4**, 3M Toxicity Study Report dated March 20, 1979, produced in the Minnesota PFAS Litigation at 3MA0059073

other regulators for decades. One of 3M's chief scientists eventually resigned over the company's failure to dedicate sufficient resources to the investigation of PFOS's harms, calling the chemical the "most onerous pollutant since PCB[.]"[2]

12. On information and belief, the remaining Defendants also knew or, at a minimum, should have known about PFOS/PFOA's toxicity and environmental hazards, including through their participation in industry trade groups formed for the purpose of lobbying regulators to protect their lucrative AFFF lines of business. They certainly must have known about the chemicals' mobility, biopersistence, and ability to bioaccumulate and biomagnify during the time periods in which the Defendants manufactured AFFF products.

13. Safer alternatives to AFFF containing or breaking down into PFOS and/or PFOA were available when Defendants designed, manufactured, and sold the products that are the subject of this Complaint. Indeed, upon regulatory urging, several of the Defendants have altered the chemical make-up of their AFFF products to rely on short-chain fluorosurfactants (six carbon atoms or fewer, compared to the eight carbon atoms in PFOA and PFOS) that they claim are less biopersistent and less toxic. Defendants could have done so much sooner.

14. Moreover, flourine-free firefighting foams that can suppress fires of flammable liquids just as effectively are available (and have in fact long been used

[2] **Ex. 5**, Email chain dated March 26, 1999, produced in the Minnesota PFAS Litigation at 3MA01373218

for that purpose by large commercial airports outside the United States) and do not pose the types of harms to the environment and human health that AFFF containing fluorinated substances does. Defendants knew or should have known about this alternative, but apparently never adequately pursued it, electing instead to protect their existing lines of AFFF business at all costs.

15. Defendants also failed to provide adequate warnings and instructions with their AFFF products, including both before and after selling such products. Defendants failed to adequately advise their customers, the public, and the State, or anyone else, about (i) the harms AFFF products containing and/or breaking down into PFOS or PFOA posed to the natural environment and human health; (ii) methods of environmentally safe disposal of Defendants' AFFF products; and (iii) designs of AFFF release sites, including firefighting training sites, that may eliminate or limit the release of PFOS and/or PFOA into the environment or otherwise mitigate their detrimental environmental effects.

16. Defendants by their conduct bear ultimate responsibility for the release of vast amounts of PFOS and/or PFOA into Ohio's natural environment, contaminating the State's waterways, waterbodies, aquifers, soils, sediments, fish and animal tissue, and biota, and threatening the health of Ohio's citizenry.

17. The State has detected dangerously high levels of PFOS and/or PFOA released into the environment from Defendants' products at or near several military

and firefighting training sites within Ohio. Additional testing will undoubtedly unearth further contamination.

18. Defendants' AFFF products containing and/or breaking down into PFOS and/or PFOA caused and will continue to cause direct injury to Ohio's public natural resources.

19. Ohio has incurred and will continue to incur significant costs in monitoring, investigating, analyzing, and remediating harms caused by the PFOS and/or PFOA released by Defendants' products into the environment and contaminating public natural resources within the State. These costs and related damages that have resulted from Defendants' conduct should rightfully be shouldered by Defendants, not Ohio's citizens.

II. JURISDICTION AND VENUE

20. The natural resources that are the subject of this suit all rest within the State of Ohio. No federal subject-matter jurisdiction exists or is invoked herein.

21. Venue is appropriate pursuant to Ohio Rules of Civil Procedure 3(C)(6) because a portion of the claim for relief arose in Lucas County. The harm created by Defendants' conduct is located throughout Ohio, including Lucas County. The property, natural resources, and injury in question includes without limitation water, wildlife, soil, and land, and submerged lands, including those

within Lucas County. Defendants' AFFF products containing and/or breaking down into PFOS or PFOA were sold and/or used in Lucas County.

III. PARTIES

A. PLAINTIFF

22. The State of Ohio, by its Attorney General Michael DeWine, brings this suit pursuant to its inherent *parens patriae* authority to remedy an injury to its "quasi-sovereign interest" in the physical and economic health and well-being of a substantial segment of its population, and pursuant to its responsibilities and authority as trustee of public natural resources.

23. Ohio enjoys *parens patriae* standing in this suit because its residents are adversely affected by the presence of PFOS and PFOA released from Defendants' products in the State's public natural resources and/or suffer loss through monetary assessments or expenditures that contribute in part to the cleanup of these chemicals.

24. The PFOS and PFOA contamination caused by Defendants' products constitutes injury to Ohio's public natural resources and to other property and waters of the State, for which Ohio seeks damages, including on behalf of itself and on behalf of its residents in its *parens patriae* capacity.

25. Ohio has a quasi-sovereign interest in and fiduciary obligation to protect its public natural resources, including soils, aquatic and submerged lands, waters, aquifers, wildlife, fish, biota, and other natural resources.

26. Ohio has a proprietary interest in protecting all property owned by the State and has an interest in remediating the contamination of its property and in preventing future contamination.

27. Ohio has spent and will continue to spend substantial sums to remediate the PFOS and/or PFOA contamination caused by Defendants' products.

28. Injury to public natural resources caused by Defendants' products has resulted in loss of public use and enjoyment of those resources. The economic value of these natural resources, as well as the cost of restoring them, is substantial.

B. DEFENDANTS

29. Defendant **3M** is a publicly traded corporation organized and existing under the laws of the state of Delaware with its principal place of business at 3M Center, St. Paul, Minnesota 55144.

30. Beginning before 1970 and until at least 2000, 3M designed, manufactured, marketed, sold, and/or distributed AFFF products containing or breaking down into PFAS, including PFOS and PFOA. 3M was the only company that designed, manufactured, marketed, sold, and/or distributed AFFF products containing or breaking down into PFOS. Upon information and belief, these 3M

products were used and released into the environment within Ohio, including at one or more of the sites discussed in this Complaint.

31. Defendant **Tyco** is a limited partnership formed and existing under the laws of the state of Delaware with its principal place of business at One Stanton St., Marinette, Wisconsin 54143. Tyco is an indirect subsidiary ultimately wholly owned by Johnson Controls International plc, an Irish public limited company listed on the New York Stock Exchange. Tyco is the successor in interest to The Ansul Company ("Ansul" and with Tyco, "Tyco/Ansul"), which beginning in or about 1976 designed, manufactured, marketed, sold, and/or distributed AFFF products containing or breaking down into PFAS, including PFOA. Following Tyco's acquisition of Ansul, Tyco/Ansul continued to design, manufacture, market, sell, and/or distribute AFFF products containing or breaking down into PFOA. Upon information and belief, these Tyco/Ansul products were used and released into the environment within Ohio, including at one or more of the sites discussed in this Complaint.

32. Defendant **Chemguard** is a corporation organized and existing under the laws of the State of Texas with its principal place of business at One Stanton St., Marinette, Wisconsin 54143. Like Tyco, Chemguard is a subsidiary of Johnson Controls International plc. Beginning in or around 1998, Chemguard designed, manufactured, marketed, sold, and/or distributed AFFF products

containing or breaking down into PFAS, including PFOA. Upon information and belief, these Chemguard products were used and released into the environment within Ohio, including at one or more of the sites discussed in this Complaint.

33. Defendant **Buckeye** is a corporation organized and existing under the laws of the State of Ohio with its principal place of business at 110 Kings Road, Kings Mountain, North Carolina 28086. Beginning in or around 2004, Buckeye designed, manufactured, marketed, sold, and/or distributed AFFF products containing or breaking down into PFAS, including PFOA. Upon information and belief, these Buckeye products were used and released into the environment within Ohio, including at one or more of the sites discussed in this Complaint.

34. Defendant **National Foam** is a corporation organized and existing under the laws of the State of Delaware with its principal place of business at 350 East Union Street, West Chester, Pennsylvania 19382. Beginning in or around 1973, National Foam designed, manufactured, marketed, sold, and/or distributed AFFF products containing or breaking down into PFAS, including PFOA. Upon information and belief, these National Foam products were used and released into the environment within Ohio, including at one or more of the sites discussed in this Complaint.

35. Defendant **Angus Fire** is a corporation organized and existing under the laws of the State of Delaware with its principal place of business at 141 Junny

Road,

Angier, North Carolina 27501. Beginning in or around 1994, Angus Fire designed, manufactured, marketed, sold, and/or distributed AFFF products containing or breaking down into PFAS, including PFOA. Upon information and belief, these Angus Fire products were used and released into the environment within Ohio, including at one or more of the sites discussed in this Complaint.

IV. FACTUAL ALLEGATIONS

A. PFOS AND PFOA ARE DANGEROUS CHEMICALS THAT THREATEN HUMAN AND ENVIRONMENTAL HEALTH AND SAFETY

1. Physical and Chemical Properties of PFOS and PFOA

36. PFAS are a group of synthetic chemical compounds containing fluorine and carbon atoms.

37. They are known as "surfactants" in that they reduce the surface tension of water.

38. As such, these chemicals have been used for decades in the manufacture of household and commercial products that resist heat, stains, oil, and water, including carpet and clothing treatments, cardboard packaging and leather products, emulsifiers, wetting agents, additives and coatings, processing aids in the manufacture of fluoropolymers such as nonstick coatings on cookware, and membranes for clothing that are both waterproof and breathable.

39. PFAS are man-made; they do not occur naturally.

40. The two most widely studied types of PFAS are PFOA and PFOS, both synthetic, fully fluorinated organic acids with eight carbon atoms.

41. PFOA and PFOS have a number of unique properties that, together, turn these chemicals into a grave threat to public health and the natural environment.

42. ***PFOA and PFOS are mobile and persistent***: once introduced, they readily spread into the natural environment where they break down very slowly, if at all.

43. The compounds are characterized by multiple carbon-fluorine bonds, which are exceptionally strong and stable. As such, they are extremely persistent in the environment and resistant to metabolic and environmental degradation.

44. PFOA and PFOS further easily dissolve in water and are thus mobile and can readily spread once in the environment. They contaminate soils and leach from the soil into groundwater, where they travel significant distances.

45. ***PFOS and PFOA bioaccumulate and biomagnify*** in the environment. Bioaccumulation occurs when an organism absorbs a substance at a rate faster than that at which the substance is lost by catabolism and excretion. Biomagnification is the increasing concentration of a substance in the tissues of tolerant organisms at successively higher levels in a food chain.

46. PFOS and PFOA are extremely stable and persistent, and as such, once ingested, they tend to bioaccumulate in individual organisms for a significant period of time.

47. For example, PFOS and PFOA have been shown to accumulate to levels of concern in fish, reaching concentrations of several thousands of times higher than in water. The compounds have been detected in both wild-caught and farmed fish, as a result of bioaccumulation and/or trophic transfer, i.e. biomagnification up the food chain.

48. PFOA has also been shown to bioaccumulate in air-breathing species, including humans.

49. PFOA and PFOS further bioaccumulate in the unborn and in infants by crossing the placenta from mother to fetus and by passing to infants through breast milk.

50. PFOA and PFOS biomagnify up the food chain—for example, when humans eat fish that have ingested PFOA or PFOS. PFOS has been observed in high concentrations in various animals higher up in the food chain, including bald eagles, walrus, narwhals, and beluga whales.

51. Finally, ***PFOS and PFOA are toxic***. Numerous studies make plain that exposure to or ingestion of these chemicals can pose serious risks to humans and to animals.

2. PFOS and PFOA are Harmful to Human Health

52. Human epidemiological studies, relied upon by the EPA for purposes of the agency's health advisories on PFOA, have found associations between PFOA exposure and high cholesterol, increased liver enzymes, decreased vaccination response, thyroid disorders, pregnancy-induced hypertension and preeclampsia, and testicular and kidney cancer.

53. Human epidemiological studies, relied upon by the EPA for purposes of the agency's health advisories on PFOS, have found associations between PFOS exposure and high cholesterol, thyroid disease, and adverse reproductive and developmental effects, including gestational diabetes, preeclampsia, and low birth weight. The developing fetus and newborns are particularly sensitive to PFOS-induced toxicity.

54. PFOS and PFOA are toxic to laboratory animals, producing reproductive, developmental and systemic effects in laboratory tests.

55. The World Health Organization's International Agency for Research on Cancer has found that PFOA is possibly carcinogenic to humans.

56. The EPA has found that there is suggestive evidence that PFOS and PFOA may cause cancer in humans.

3. PFOS/PFOA Are Widespread Contaminants, But Regulators' (and the Public's) Understanding of Levels of Acceptably Safe Exposures To These Chemicals Continues To Evolve

57. Given their physical and chemical properties, PFOS and PFOA have become incredibly widespread in the environment, posing an environmental and human health crisis in Ohio and beyond.

58. Indeed, PFOS and PFOA have been detected in environmental media and biota in many parts of the world, including oceans and the Arctic.

59. The chemicals have been found in cereals, fish, soft drinks, milk, olive oil, and meat, as well as in prepared foods.

60. According to the EPA, PFOA and PFOS have been detected in the blood serum of 99% of the U.S. population. This is particularly troubling given the real and significant adverse health effects these chemicals pose.

61. The Director of the U.S. Center for Disease Control's National Center for Environmental Health, Patrick Breysse, reportedly described the chemicals in October of 2017 as "one of the most seminal public health challenges for the next decades."

62. This understanding of PFAS contamination as a widespread public health crisis has been slow to evolve, however, and has only fairly recently garnered broad attention. Indeed, although the EPA began to investigate the safety of PFOA and PFOS in or around 1998 following some limited disclosures by 3M and others, the agency did not begin to issue health advisories for these chemicals until January 8, 2009.

63. The 2009 EPA health advisory noted merely that "action should be taken to reduce exposure" to drinking water containing levels of PFOA and PFOS exceeding 400 parts per trillion ("ppt") and 200 ppt, respectively.

64. In May 2016, the EPA significantly revised its PFOA and PFOS health advisory, recommending that drinking water concentrations for PFOA and PFOS, either singly or combined, should not exceed 70 ppt.

65. Notably, the EPA's health advisories are only "informal technical guidance to assist federal, state and local officials, as well as managers of public or community water systems in protecting public health. They are not regulations and should not be construed as legally enforceable federal standards."

66. Despite the purported stringency of EPA's newly-announced guidelines, recently proposed and adopted state standards suggest they may still not be strict enough.

67. Although calls for a national, legally enforceable standard for PFAS contamination are growing louder, none has been passed yet.

B. DEFENDANTS' AFFF PRODUCTS HAVE FOR DECADES CONTAMINATED THE ENVIRONMENT WITH PFOS AND PFOA

68. The PFAS application critical to the claims asserted in this Complaint is AFFF, which is widely used to suppress and extinguish fires of flammable liquids, such as fuel and oil.

69. In the 1940s, 3M began to experiment with a process called electrochemical fluorination to create the carbon-fluorine bonds that are the key components of PFAS, including PFOA and PFOS.

70. The electrochemical fluorination process used by 3M results in both PFOA and PFOS.

71. The other major carbon-flourine bond producing process, which was used in the manufacture of the surfactants that the remaining Defendants used in their production of AFFF products, is called telomerization. This process results in PFOA, but not PFOS.

72. Recognizing the compounds' strong surfactant properties described above and building on its earlier experiments, 3M began to develop AFFF containing PFOS in the early 1960s to suppress flammable liquid fires that cannot be effectively extinguished with water alone.

73. 3M is the only AFFF manufacturer that used electrochemical fluorination to create carbon-fluorine bonds. 3M thus produced the only AFFF that contained PFOS.

74. Formulations of AFFF that were manufactured by the Defendants other than 3M utilized surfactants produced through telomerization and contain or break down into PFOA.

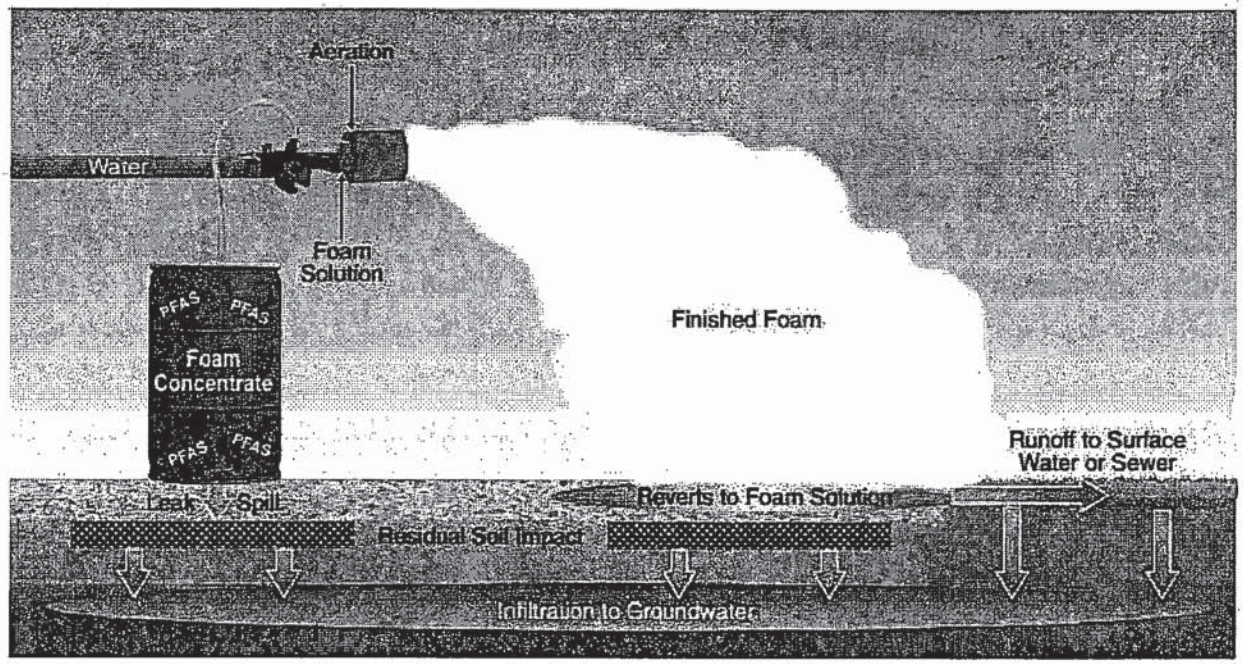
75. In the late 1960s, the United States military issued military specification MIL-F-24385 governing the requirements for AFFF ("AFFF Mil-Spec"). It requires that the AFFF concentrate "consist of fluorocarbon surfactants plus other compounds . . . ." The AFFF Mil-Spec, however, contains no further requirements concerning these fluorocarbons surfactants, such as the length of the fluorine-carbon chain.

76. The AFFF Mil-Spec also states that "[t]he material shall have no adverse effect on the health of personnel when used for its intended purpose." The current version of the AFFF Mil-Spec still contains that language.

77. From the 1960s to about 1973, 3M was the sole supplier of AFFFs.

78. Beginning in 1973, flourotelomer-based AFFF manufacturers entered the market and became AFFF suppliers to the military and others—first National Foam in or about 1973, followed by Tyco/Ansul (~1976), Angus Fire (~1994), Chemguard (~1998), and Buckeye (~2004).

79. AFFF is applied by firefighters in the field by mixing foam concentrate and water to make a foam solution. When applied to a fire, the foam solution is aerated at the nozzle. The foam solution is sprayed out to coat the fire, blocking the supply of oxygen feeding the fire and creating a cooling effect and evaporation barrier. A film also forms to smother the fire after the foam has dissipated:



80. In other words, it is intended by, and foreseeable to, the Defendants that AFFF will be mixed with water and sprayed in such a manner that it can freely seep into the groundwater and soil and contaminate the environment, unless precautionary measures are taken to prevent its introduction into the environment.

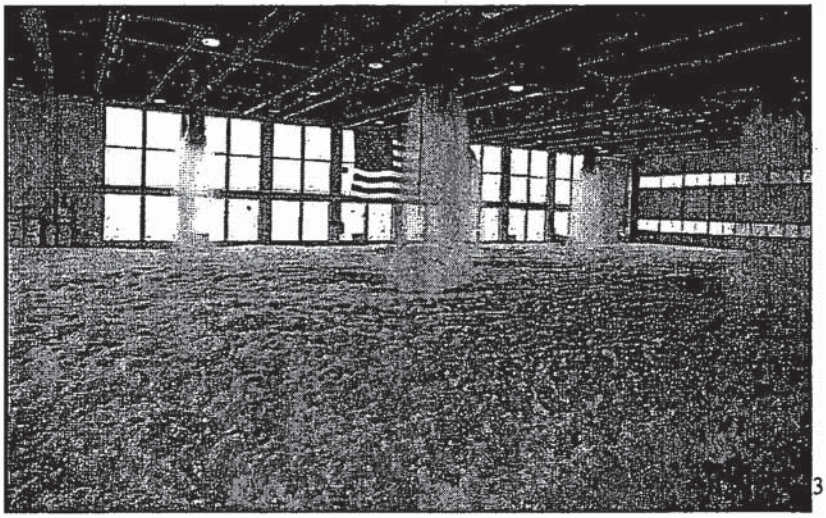
81. On information and belief, AFFF containing and/or breaking down into PFOA and/or PFOS is the only major PFOA/PFOS application that, when used as intended, releases these toxic chemicals directly into the environment in a manner enabling them to freely seep into the groundwater – potentially contaminating drinking water supplies – and travel long distances to cause further, widespread environmental contamination.

82. A single firefighting event or training exercise may result in the release of thousands of gallons of foam solution laced with PFOS or PFOA that then enter and contaminate the environment.

83. For decades, AFFF products containing and/or breaking down into PFOS and/or PFOA have been stored and used for fire suppression, fire training, and flammable vapor suppression at hundreds of military installations and civilian airports, as well as at petroleum refineries and storage facilities, and chemical manufacturing plants throughout the United States, including in Ohio.

84. Additionally, local fire departments in numerous communities have used and maintained quantities of AFFF in their inventories.

85. Fire training exercises and fire suppression systems testing involving AFFF are common, particularly on military installations, and have been performed many thousands of times since the 1960s, each time releasing vast quantities of toxic chemicals into the environment. The following images, reprinted as part of the investigative series "The Teflon Toxin" published by journalists at The Intercept, depict firefighting training exercises/suppression system testing, drenching the test space in AFFF.



[3]

86. AFFF use has been identified as one of the main contributors to the widespread environmental contamination with PFOA and PFOS.

87. Despite the recent phase-out of longer-chain PFAS, much of the current AFFF stockpiles still contain long-chain PFAS constituents due to the long

[3] *See* The Intercept, *Poisoning the Well*, available at https://theintercept.com/2015/12/16/toxic-firefighting-foam-has-contaminated-u-s-drinking-water-with-pfcs/; The Intercept, *The U.S. Military is Spending Millions to Replace Toxic Firefighting Foam with Toxic Firefighting Foam*, available at https://theintercept.com/2018/02/10/firefighting-foam-afff-pfos-pfoa-epa/

shelf-life of these products. AFFF containing or breaking down into PFOS and/or PFOA thus continues to be widely stored and used, including in Ohio.

88. Significantly, recognizing the dangers of PFOA and PFOS, as of 2017 the AFFF Mil-Spec has been amended to state that the Department of Defense seeks "to acquire and use a non-fluorinated AFFF formulation or equivalent firefighting agent to meet [its] performance requirements …"

89. Had Defendants been forthright about their products' chemical properties and the environmental and human health hazards they posed, the Department of Defense (and regulatory agencies) would have taken steps to prevent, control, or minimize the environmental and human health threats from AFFF containing and/or breaking down into PFOA of PFOS much sooner, or would never have used them in the first place.

C. THE DEFENDANTS KNEW ABOUT BUT CONCEALED THE DANGERS OF PFOS AND/OR PFOA CONTAINED IN AFFF

90. As revealed in connection with the State of Minnesota's litigation against 3M concerning PFAS contamination (the "Minnesota PFAS Litigation"),[4] 3M has known for decades that PFOA and PFOS are mobile and persistent, bioaccumulative and biomagnifying, and toxic to human and animal life.

[4] *State of Minnesota v. 3M Company*, Case No. 27-cv-10-28862 (District Court, Fourth Judicial District, Hennepin County, Minnesota).

91. Upon information and belief, the other Defendants, each of which manufactured AFFF containing or breaking down into PFOA, likewise knew of the dangers to human and environmental health posed by PFOA, including through information they obtained as part of their participation in trade industry associations.

92. All Defendants were careful to withhold the most damning information about PFOS and/or PFOA from their customers, the public, and regulators.

1. 3M Knew About but Concealed the Dangers of PFOS and PFOA

93. 3M conducted extensive toxicity studies on PFAS, including PFOS and PFOA, as early as the 1950s, concluding that the chemicals were toxic.

94. Further toxicity studies conducted by 3M scientists in the late 1970s confirmed that the chemicals were even "more toxic than anticipated."[5]

95. In 1978, 3M conducted studies on monkeys and rats, feeding them various dosages of PFOS and PFOA. All monkeys in the study died within the first few days after being given PFOS at a dosage of 4.5 mg/kg/day.[6] Monkeys being given 100 mg/kg/day of PFOA "all died during weeks 2 through 5 of the

[5] **Ex. 4**, 3M Toxicity Study Report dated March 20, 1979, produced in the Minnesota PFAS Litigation at 3MA0059073.

[6] **Ex. 2**, 3M Central Analytical Laboratory Report dated August 4, 1978, produced in the Minnesota PFAS Litigation at 3M_MN02343995.

study."[7] The company's studies showed that both PFOA and PFOS affected the liver and gastrointestinal tract of the species tested.[8]

96. 3M concluded that PFOS was "the most toxic" of the compounds studied and "certainly more toxic than anticipated."[9]

97. 3M consulted with Harold Hodge, a well-known toxicologist, who emphasized that it was of "utmost importance" to determine whether these chemicals "or its metabolites are present in man, what level they are present, and the degree of persistence (half-life) of these materials."[10]

98. Further, in 1975, 3M was alerted by third-party researchers that PFOS was detectable in human blood serum and thus had obviously spread beyond the immediate site of its applications and was bioacccumulating.

99. 3M's own research confirmed by the next year that the level of fluorochemicals in the blood of its own workers was "1,000 TIMES NORMAL."[11]

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[7] **Ex. 3**, 3M Toxicity Study Report dated November 10, 1978.

[8] **Ex. 4**, 3M Toxicity Study Report dated March 20, 1979, produced in the Minnesota PFAS Litigation at 3MA0059073.

[9] *Id.*

[10] **Ex. 6**, 3M Meeting Minutes dated June 7, 1979, produced in the Minnesota PFAS Litigation at 3MA00592803.

[11] **Ex. 1**, "Organic Fluorine Compounds in Blood Chronology" dated October 19, 1977, produced in the Minnesota PFAS Litigation at 3M_MN00000479 at 481.

100. Conducting research around its manufacturing plants, 3M knew by 1979 that its fluorochemicals "bioaccumulated more readily in the gastrointestinal tract, fat and reproductive system [at least in] channel catfish[.]"[12]

101. By 1979, 3M recognized that fluorochemicals may pose a cancer risk. Indeed, one of its scientists pressed that it was "paramount to begin now an assessment of the potential (if any) of long term (carcinogenic) effects for these compounds which are known to persist for a long time in the body and thereby give long term chronic exposure."[13]

102. 3M nonetheless continued to assure its customers, for example the U.S. Navy, a major purchaser of 3M's AFFF products (which, by the mid-1970s, had raised concerns about the environmental impact of AFFF releases into the environment), that its products were "biodegradable and will have no adverse effects on the environment."[14] That assurance was knowingly false.

103. 3M never published its toxicity studies and worked actively to stifle research on the adverse effects of PFOA and PFOS.

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[12] **Ex. 7**, 3M Technical Report Summary dated May 22, 1979, produced in the Minnesota PFAS Litigation at 3MA01409559.

[13] **Ex. 8**, 3M Interoffice Correspondence dated July 6, 1979, produced in the Minnesota PFAS Litigation at 3MA00593079.

[14] **Ex. 9**, February 1974 Report by the Naval Research Laboratory titled "A Substitute Liquid for AFFF (Aqueous Film Forming Foam) Concentrate for Checking Proportioners" citing a Letter from 3M to Navmat, dated Feb. 29, 1972.

104. Indeed, according to evidence developed during the Minnesota PFAS Litigation, 3M kept John Giesy, Ph.D., Professor and Canada Research Chair in Environmental Toxicology in the Department of Veterinary Biomedical Sciences and Toxicology Centre at the University of Saskatchewan, on its payroll to the tune of millions of dollars for the purpose of influencing independent academic research. It was Prof. Giesy's professed goal to keep unfavorable papers regarding PFAS out of the academic literature.

105. 3M also advised its employees not to put their thoughts and research concerning PFOS or PFOA to writing, lest such communications would need to be disclosed during discovery in likely litigation.[15]

106. 3M also knew full well the environmental implications associated with PFOS and PFOA but refused to allow testing to perform precise ecological risk assessments.

107. One of 3M's longtime scientists, Dr. Richard Purdy, stated in an internal email: "PFOS is the most onerous pollutant since PCB and you want to avoid collecting data that indicates that it is probably worse. I am outrage[d.]"[16]

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[15] **Ex. 10**, March 28, 1999 resignation letter by 3M scientist Richard Purdy, produced in the Minnesota PFAS Litigation at 3MA00480715 at 16 ("3M told those of us working on the fluorochemical project not to write down our thoughts or have email discussions on issues because of how our speculations could be viewed in a legal discovery process.").

[16] **Ex. 5**, Email chain dated March 26, 1999, produced in the Minnesota PFAS Litigation at 3MA01373219.

108. Despite 3M's knowledge of PFOS and PFOA toxicity and potential carcinogenicity, its mobility and persistence in the environment, and its tendency to bioaccumulate and biomagnify, the company continued to manufacture, sell, and distribute AFFF containing and/or breaking down into these chemicals until at least 2000.

109. Dr. Purdy resigned shortly thereafter, exhausted by the company's "roadblocks, delays, and indecision" concerning research on PFAS' environmental effects and failure to address their known environmental harms:

- **3M continues to make and sell these chemicals, though the company knows of an ecological risk assessment I did that indicates there is a better than 100% probability that perfluorooctansulfonate is biomagnifying in the food chain and harming sea mammals. This chemical is more stable than many rocks. And the chemicals the company is considering for replacement are just as stable and biologically available. The risk assessment I performed was simple, and not worst case. If worst case is used, the probability of harm exceeds 100,000%.**

Dr. Purdy concluded that he could no longer work for a company "concerned with markets, legal defensibility and image over environmental safety."[17]

110. Dr. Purdy copied the EPA on his March 1999 resignation letter.

111. Shortly thereafter, 3M supplemented its prior submissions to the EPA with critical information referenced by Dr. Purdy. In 2000, 3M ceased production of PFOS and PFOA.

[17] **Ex. 10**, March 28, 1999 resignation letter by 3M scientist Richard Purdy.

112. In April 2006, 3M paid a penalty of more than $1.5 million to the EPA for its failure to disclose pertinent studies regarding PFOA and PFOS.

2. All Defendants Were Aware of The Harmful Effects of AFFF, but Were Intent On Protecting Their Existing Business To The Detriment of the Public

113. On information and belief, Tyco/Ansul, Chemguard, Buckeye, National Foam, and Angus Fire also knew or, at a minimum, should have known that in its intended and common use, AFFF containing or breaking down into PFOA would injure the natural environment and threaten public health.

114. Additionally, all Defendants knew or, at a minimum, should have known that their AFFF products containing or breaking down into PFOS or PFOA, given their chemical composition, easily dissolve in water (and the products were designed to be mixed with water), are mobile, resist degradation, and tend to bioaccumulate and biomagnify.

115. On information and belief, this information was accessible to each of the Defendants because each is an expert in the field of AFFF manufacture with understanding of and substantial information on the chemical compounds forming part of their respective AFFF products.

116. On information and belief, this information was also accessible to each of them as part of their ongoing involvement in various trade associations and groups formed for the purpose of defending the AFFF franchise.

117. One such group, the Firefighting Foam Coalition ("FFFC"), was formed in 2001 to dispel concerns the EPA had raised about AFFF's environmental viability.

118. All of the Defendants other than 3M were members of the FFFC during times relevant to the claims in this Complaint, and at least Tyco/Ansul, Chemguard, National Foam, and Angus Fire are current FFFC members.

119. E.I. DuPont de Nemours and Co. ("DuPont"), which manufactured fluorinated compounds containing or breaking down into PFOA specifically for use in the manufacture of AFFF, but did not manufacture AFFF, was a founding member of the FFFC.

120. DuPont had long known about PFOA's toxicity, persistence, and tendency to bioaccumulate and biomagnify. By 1961, DuPont's own researchers had concluded that PFOA was toxic and should be "handled with extreme care" and a few years later, DuPont had knowledge that PFOA caused adverse liver reactions in dogs and rats. By 1976, DuPont was also aware of research reports that detected organic flourine in blood bank samples in the U.S., which the researchers believed to be a potential result of human exposure to PFOA. Through the decades, DuPont had access to mounting evidence of PFOA's toxicity and negative impact on the environment, but failed to disclose this information to regulatory agencies and the public at large.

121. Through the FFFC, Defendants and DuPont worked together closely to protect AFFF from regulatory scrutiny. This close cooperation, including with respect to messaging on PFOA's toxicological profile, strongly suggests that DuPont shared with or made available to the Defendant FFFC members non-public information concerning PFOA's properties, including its toxicity, persistence, and bioaccumulativity during their joint effort to shield their respective, lucrative AFFF lines of business in the face of their products' foreseeable deleterious impact on natural resources and human health.

122. The FFFC lobbied hard for AFFF. The organization regularly published newsletters concerning the viability of telomer-based firefighting foam and had its members attend conferences, all with the express purpose of assuaging worries about the environmental concerns circling AFFF and downplaying the obvious benefits of AFFF alternatives, such as fluorine free foam.

123. At an August 2002 conference in Manchester, England, for example, Steve Korzeniowski of DuPont "presented the latest scientific information on telomer-based products, including the fluorosurfactants used in AFFF firefighting agents." Korzeniowksi discussed the toxicology of PFOS and PFOA, emphasizing that 3M had phased out PFOS-containing AFFF, but that the Defendants continued to produce telomer-based AFFF, which was, purportedly, safer than 3M's products.

124. The other FFFC members, including Defendants, repeated those messages relentlessly. Indeed, in 2003, the FFFC's member companies presented the same narrative at the Workshop on Fire Suppression Technologies in Mobile, Alabama, and the NFPA World Safety Conference and Exposition in Dallas, Texas, among other events.

125. Over many years, at conferences throughout the world, in journals, and in meetings with the U.S. military and the EPA, the FFFC repeated this key talking point over and over: Only one PFAS chemical, PFOS, had been taken off the market. Since the FFFC members' products did not contain PFOS, their products were safe.

126. The FFFC members' key message on telomer-based AFFF was knowingly false. Each of the FFFC members' AFFF products contained or broke down into PFOA, which they knew or, at a minimum, should have known was equally harmful to the environment and public health as was PFOS.

127. The Defendants, other than 3M, eventually transitioned to the use of short-chain fluorotelomers with a maximum of six carbon atoms, claiming those chemicals are safer to environmental and human health than the long-chain compounds.

128. Defendants could have begun to transition from long-chain to short-chain fluorotelomers much earlier.

129. Their failure to avail themselves of what they claim is a feasible alternative to the AFFF products containing or breaking down into PFOA that they previously manufactured and sold, and which substantially mitigates the risk of human and environmental harm from AFFF products, only confirms that their AFFF products containing long-chain fluorotelomers were not reasonably safe for their intended uses.

130. What is more, effective fluorine-free firefighting foams that do not pose the same risks to human health and the environment as Defendants' products exist and are used in some of the world's largest airports, including London Heathrow, London Gatwick, Copenhagen, Stuttgart and Dubai, amongst others. All 27 of Australia's airports have been using fluorine-free foams for many years.

131. Indeed, leading fire safety and regulatory experts have opined that there are simply no justifications for continued use of toxic foams given this successful, widespread use of the environmentally safe alternative.

132. According to a report issued by a panel of experts of IPEN, a global network of public interest NGOs dedicated to the reduction of toxic chemicals, fluorine-free firefighting (F3) foams are viable alternatives to fluorinated AFFF and comparable by all measures. But unlike fluorinated foams, F3 foams do not pollute the environment indefinitely, or put human or animal health at risk; there is no expensive clean up; remediation costs are negligible or zero; and there are no

significant legal and financial liabilities. Public health values such as clean drinking water are not compromised, and, finally, there is no erosion of public confidence in political institutions and government agencies.

133. As of 2017, the AFFF Mil-Spec has been amended to state that the Department of Defense seeks "to acquire and use a non-fluorinated AFFF formulation or equivalent firefighting agent to meet [its] performance requirements…."

134. Had Defendants been forthright about their AFFF products' chemical properties and the environmental and human health hazards they posed, the Department of Defense (and other AFFF users) would have sought to replace existing AFFF with fluorine free firefighting foam, used successfully abroad, much sooner.

135. Defendants failed to adequately research and investigate the design, manufacture, or sale of fluorine-free firefighting foam, or did so and concealed their results. They avoided fluorine-free alternatives to protect their existing, lucrative AFFF lines of business.

136. Defendants' failure to pursue this feasible alternative to AFFF containing or breaking down into PFOS or PFOA further confirms that their AFFF products were not reasonably safe for their intended uses.

D. DEFENDANTS' AFFF PRODUCTS HAVE CAUSED (AND CONTINUE TO CAUSE) WIDESPREAD ENVIRONMENTAL CONTAMINATION WITH PFOS AND PFOA IN OHIO

137. Defendants' AFFF products containing or breaking down into PFOS/PFOA have been used for decades throughout Ohio military bases, civilian airports, firefighting training centers, and other facilities.

138. Thus far, PFOS/PFOA contamination caused by the use of Defendants' AFFF products has been detected in several locations in Ohio and the scope of detected contamination is likely to increase with additional testing.

139. The United States Air Force stored AFFF products containing PFOA or PFOS and used and discharged them at the following sites within Ohio (the following list is not exhaustive):

a. Air Force Plant 85, Columbus, Ohio (Franklin County);

b. Mansfield-Lahm Regional Airport, Mansfield, Ohio (Richland County);

c. Newark Air Force Base (former), Heath, Ohio (Licking County);

d. Springfield-Beckley Municipal Airport, Springfield, Ohio (Clark County);

e. Toledo Express Airport, Swanton, Ohio (Lucas County);

f. Wright-Patterson Air Force Base, Dayton, Ohio (Montgomery County); and

g. Youngstown-Warren Air Force Base near Youngstown and Warren, Ohio (Trumbull County).

140. Defendants manufactured, sold, and/or distributed AFFF products containing or breaking down into PFOS and/or PFOA that were used at these and other sites in Ohio.

141. Defendants' AFFF products containing or breaking down into PFOA and/or PFOS were also used in Ohio civilian airports and firefighting facilities, including at the Newport Volunteer Fire Department in Newport, Ohio (Washington County).

142. During firefighting and firefighting training exercises at these and other Ohio sites, firefighters sprayed AFFF containing or breaking down into PFOS or PFOA, per its intended use, directly on or near the ground, caused it to be disposed, and spilled it or otherwise caused it to be discharged or released into the environment.

143. Additional discharges and releases may have occurred in connection with storage and handling of AFFF. These activities resulted in foreseeable discharges or releases of PFOA and/or PFOS from Defendants' AFFF products into nearby groundwater, soil, and other environmental media.

144. In short, the normal, intended, and foreseeable manner of storage, use, and disposal of Defendants' AFFF products directly resulted in the introduction of PFOA and/or PFOS into Ohio's waters, soils, and other natural resources.

145. Upon information and belief, AFFF products containing or breaking down into PFOS or PFOA manufactured by each Defendant were discharged or released into the environment at or from these sites.

146. Upon information and belief, the instructions, labels and/or material safety data sheets that Defendants provided with their AFFF products, if any, during the times relevant to the claims in this Complaint did not fully or sufficiently describe the human and animal health and environmental hazards of AFFF about which Defendants knew or should have known.

147. Upon information and belief, the instructions, labels and/or material safety data sheets that Defendants provided with their AFFF products, if any, during the times relevant to the claims in this Complaint did not provide appropriate warnings and instructions concerning the environmentally safe disposal of AFFF that were known or should have been known to Defendants.

148. Upon information and belief, the instructions, labels and/or material safety data sheets that Defendants provided with their AFFF products, if any, during the times relevant to the claims in this Complaint did not provide appropriate warnings and instructions concerning the risks that, when used and/or

disposed of as intended, chemicals contained in AFFF, including PFOS and/or PFOA, would enter the environment, including by seeping into the groundwater, cause further contamination of environmental media at great distances, would not degrade, and would eventually bioaccumulate and biomagnify in animal tissue, even though these risks were known or should have been known to Defendants.

149. Upon information and belief, the instructions, labels and/or material safety data sheets that Defendants provided with their AFFF products, if any, during the times relevant to the claims in this Complaint did not provide appropriate instructions regarding precautions that must be taken at firefighting test-sites in a manner that would potentially eliminate or limit the release of PFOA and/or PFOS into the environment, even though the hazards of failing to appropriately contain PFOA and/or PFOS were known or should have been known to Defendants.

150. For example, instructions to install a liner under a testing area or outfitting area test-sites with appropriate water filtration systems could have significantly contained the spread of PFOS/PFOA into the environment. Defendants knew this, but failed to warn or instruct anyone that their products should only be stored, used, and disposed in conjunction with an effective liner or catch basin, or water filtration system capable of removing PFOS and PFOA.

151. Upon information and belief, the instructions, labels and/or material safety data sheets that Defendants provided with their AFFF products during the times relevant to the claims in this Complaint, if any, did not provide appropriate warnings of potential groundwater pollution through PFOA and/or PFOS nor did they advise the AFFF user to install appropriate water filtration devices to protect Ohio's natural resources, even though Defendants knew or should have known about the inevitability of groundwater and soil contamination through their AFFF products and consequent adverse effects in the absence of such measures.

152. Upon information and belief, sampling of groundwater, surface water, and soil near all of these sites within Ohio shows contamination by PFOS and/or PFOA, substances that were components in Defendants' AFFF products.

153. Following testing in May 2016, the Wright-Patterson Air Force Base detected PFOA/PFOS levels of 200 ppt and 700 ppt, respectively, in two on-base production wells approximately two-and-a-half miles from Huffman Dam near Dayton, Ohio. These wells serve part of the Wright-Patterson Air Force Base and the Wright-Patterson Air Force Base immediately shut down the production well with detected PFOA/PFOS concentrations of 700 ppt. Upon announcement of the EPA's May 2016 health advisory, recommending that drinking water concentrations for PFOA and PFOS, either singly or combined, should not exceed 70 ppt, Ohio EPA ordered the Wright-Patterson Air Force Base to also shut down

the second production well. Detections of PFOA/PFOS in other Wright-Patterson Air Force Base production wells range from non-detect to 48 ppt PFOA/PFOS

154. Following detections of PFOA/PFOS in early warning monitoring wells downgradient from the Wright-Patterson Air Force Base, the City of Dayton voluntarily stopped using six production wells in their Huffman Dam wellfield. After detecting PFOA/PFOS in a nearby monitoring well, the City of Dayton also stopped using an additional production well in the Mad River wellfield.

155. In February 2018, the City of Dayton informed both Ohio EPA and the Wright-Patterson Air Force Base that the City had operated a fire training center at the western edge of the Mad River Wellfield. The City provided data from area monitoring wells and soil borings, confirming PFOS contamination. As a precaution, the City of Dayton had previously stopped using five production wells closest to its fire training center. To date, these production wells continue to be offline.

156. In March 2018, the City of Dayton confirmed that PFOS/PFOA had also been detected in the City's finished water from the Ottawa Plant.

157. During testing conducted between September 2016 through about April 2017, the Ohio EPA further detected PFOA in seven private wells at or near

the Toledo Air National Guard Base, with one exhibiting PFOA at a level of 349 ppt.

158. The Newport Volunteer Fire Department in Newport, Ohio stored AFFF products containing or breaking down into PFOA or PFOS and used and discharged these products during fire training exercises it hosted for Ohio fire departments from or about 1964 to 1974. The Ohio EPA sampled the Newport site for PFOS and PFOA in October 2016 and detected PFOS at 175 ppt at the recovery well on the site.

159. The State's sampling activities to detect PFOS/PFOA from AFFF in public water supplies and other natural resources, including in or around military bases and other firefighting training sites, are ongoing.

160. As the State continues its investigation, it may discover other sites that will require remediation or restoration due to contamination with PFOA and/or PFOS from AFFF.

161. The State may also discover that further natural resources have been damaged due to such contamination.

162. Ohio has already invested significant sums in a variety of general and site-specific efforts to assess, investigate, strategize, and implement remediation plans designed to remove PFOA and/or PFOS from Ohio natural resources.

163. Ohio and its citizenry has suffered loss of use of Ohio natural resources, including certain drinking water supplies in or around the Wright-Patterson Air Force Base near Dayton, in or around the Toledo Air National Guard Base in Toledo, and likely in other locations within Ohio, and catching, selling, and/or consuming fish within or from impaired or contaminated Ohio waters, among other injuries.

V. CAUSES OF ACTION

FIRST CAUSE OF ACTION
PUBLIC TRUST DOCTRINE

164. Ohio realleges and incorporates the allegations set forth in paragraphs 1 through 163 as if fully stated herein.

165. Ohio asserts this cause of action in its capacity as trustee of a public trust.

166. Ohio is the trustee of a public trust the corpus of which comprises all public natural resources within the State of Ohio, including all public waters, soils, lands and submerged lands, wildlife, and biota.

167. In its capacity as trustee, Ohio holds all public waters, soils, lands and submerged lands, wildlife, and biota in trust for the benefit of all Ohioans. Ohio public trust law affords protection to natural resources as far as necessary to accommodate the public uses to which they might be adapted.

168. As trustee, Ohio has a fiduciary obligation to defend the corpus of this trust, including by asserting claims for damages against persons who injure the corpus of the trust.

169. Ohio law affords the State, through its Attorney General, the right to sue under the public trust doctrine in furtherance of this fiduciary obligation. This cause of action is asserted pursuant to this right.

170. Defendants manufactured, distributed, marketed, promoted, and sold AFFF products containing or breaking down into PFOS or PFOA in a manner that created hazards to human and environmental health, including the natural resources alleged above, within the State of Ohio.

171. Defendants knew, or in the exercise of reasonable care should have known, that the AFFF products containing or breaking down into PFOS or PFOA that they manufactured, distributed, marketed, promoted, and sold would end up contaminating Ohio's public natural resources, including waterways, waterbodies, aquifers, groundwater, surface water, soils, sediments, fish and animal tissue, and biota, if those products were stored, used, and disposed as intended.

172. Defendants' conduct and the presence of PFOS and/or PFOA released from Defendants' products have resulted in the impairment and/or contamination of Ohio public natural resources, including those natural resources identified hereinabove.

173. Defendants' conduct and the presence of PFOS and/or PFOA released from Defendants' products have resulted in the loss of use of Ohio public natural resources, including those natural resources identified hereinabove.

174. Defendants' conduct and the presence of PFOS and/or PFOA released from Defendants' products have resulted in degradation or elimination of the health, ecological, and other beneficial uses of Ohio public natural resources, including those natural resources identified hereinabove.

175. Defendants' conduct and the presence of PFOS and/or PFOA released from Defendants' products in Ohio public natural resources are injurious to human, animal, and environmental health.

176. Ohio suffered and continues to suffer damage from Defendants' conduct and the presence of PFOS and/or PFOA released from Defendants' products in Ohio public natural resources, including without limitation costs to assess, investigate, monitor, analyze, and remove PFOS and/or PFOA that have invaded public natural resources, to prevent PFOS and/or PFOA from injuring additional public natural resources, and to restore public natural resources whose use has been lost.

177. As a direct and proximate result of Defendants' conduct, Ohio public natural resources have been contaminated and/or impaired, and their beneficial uses have been degraded or eliminated.

178. As a further direct and proximate result of Defendants' conduct, Ohio, in its capacity as trustee over its public natural resources, has suffered and continues to suffer monetary losses in amounts to be proven at trial.

179. Defendants are strictly, jointly and severally liable for all such damages, and the State is entitled to recover all such damages and other relief as set forth below.

SECOND CAUSE OF ACTION
DESIGN DEFECT

180. Ohio realleges and incorporates the allegations set forth in paragraphs 1 through 163 as if fully stated herein.

181. Ohio asserts this cause of action pursuant to its role as public trustee of natural resources and its inherent *parens patriae* authority to defend a quasi-sovereign interest, and does not here assert or usurp claims on behalf of any individual or non-State entity harmed in his or her person or property by Defendants' conduct.

182. Defendants' AFFF products containing or breaking down into PFOS and/or PFOA were not reasonably safe as designed at the time they left Defendants' control.

183. Defendants' AFFF products' toxicity, inability to be contained once used as intended, tendency to bioaccumulate and biomagnify, and environmental persistence rendered them unreasonably dangerous at all times.

184. Defendants' AFFF products containing or breaking down into PFOS and/or PFOA were unsafe as designed, as demonstrated by numerous studies alleged hereinabove.

185. Due to their toxicity, inability to be contained once used as intended, tendency to bioaccumulate and biomagnify, and persistence, Defendants knew their AFFF products containing or breaking down into PFOS and/or PFOA were not safe at the time of manufacture because it was certain that the product would contaminate natural resources within the United States, including Ohio, and cause toxic contamination of Ohio public natural resources, including those natural resources identified hereinabove.

186. Defendants knew their AFFF products containing or breaking down into PFOS and/or PFOA were unsafe to an extent beyond that which would be contemplated by an ordinary person because of the information and evidence available to Defendants associating PFOS and PFOA exposure with adverse human and animal health effects as well as the overwhelming seriousness of creating extensive contamination of the natural environment.

187. Practical and feasible alternative designs capable of reducing the State's injuries were available. Such alternatives include, by Defendants' own representations, reformulated AFFF containing shorter-chain fluorosurfactants, as well as fluorine-free firefighting foam (F3), which is already widely and

effectively being used outside of the United States. Such alternative chemical formulations would have materially decreased the environmental persistence and toxicity of Defendants' AFFF products without eliminating their typical applications or utilities.

188. Defendants' conduct and the presence of PFOS and/or PFOA in Ohio caused and continue to cause injury to the physical and economic health and well-being of Ohio citizens.

189. Ohio has suffered and will continue to suffer damages to its public natural resources and public fisc as a result of Defendants' conduct and the presence of PFOS and/or PFOA released from Defendants' products within the State.

190. Defendants are strictly, jointly and severally liable for all such damages, and the State is entitled to recover all such damages and other relief as set forth below.

THIRD CAUSE OF ACTION
FAILURE TO WARN AND INSTRUCT

191. Ohio realleges and incorporates the allegations set forth in paragraphs 1 through 163 as if fully stated herein.

192. Ohio asserts this cause of action pursuant to its role as public trustee of natural resources and its inherent *parens patriae* authority to defend a quasi-sovereign interest, and does not here assert or usurp claims on behalf of any

individual or non-State entity harmed in his or her person or property by Defendants' conduct.

193. Defendants' AFFF products containing or breaking down into PFOS and/or PFOA were not reasonably safe at the time they left Defendants' control because they lacked adequate warnings.

194. At the time Defendants manufactured, distributed, marketed, promoted, and sold AFFF products containing or breaking down into PFOS and/or PFOA, they knew their products were not safe because it was certain that PFOS and/or PFOA would contaminate natural resources within the United States, including Ohio, and cause toxic contamination of Ohio public natural resources, if those products were used as intended.

195. Despite Defendants' knowledge of the attendant risks, Defendants failed to provide adequate warnings and instructions that their products, if used as intended, would adversely affect the natural environment and human health.

196. Despite Defendants' knowledge of the attendant risks, Defendants failed to provide adequate warnings and instructions that their products, if used as intended, would contaminate Ohio public natural resources with toxic materials harmful to the environment, wildlife, and human health.

197. Despite Defendants' knowledge of the attendant risks, Defendants failed to provide adequate warnings and instructions concerning the environmentally safe disposal of their products.

198. Despite Defendants' knowledge of the attendant risks, Defendants failed to provide adequate warnings and instructions concerning the precautions that must be taken at firefighting test-sites and other foreseeable sites at which their AFFF products would be used, in order to eliminate or limit the release of PFOA and/or PFOS into the environment.

199. Defendants continued to conceal the dangers of AFFF products containing or breaking down into PFOS and/or PFOA after they manufactured, distributed, marketed, promoted, and sold such products.

200. Without adequate warnings or instructions, Defendants' products were unsafe to an extent beyond that which would be contemplated by an ordinary person.

201. Defendants' conduct and the presence of PFOS and/or PFOA released by Defendants' products in Ohio caused and continue to cause injury to the physical and economic health and well-being of Ohio citizens.

202. Ohio has suffered and will continue to suffer damages to its public natural resources and public fisc as a result of Defendants' conduct and the presence of PFOS and/or PFOA released by Defendants' products within the State.

203. Defendants are strictly, jointly and severally liable for all such damages, and the State is entitled to recover all such damages and other relief as set forth below.

FOURTH CAUSE OF ACTION
NEGLIGENCE

204. Ohio realleges and incorporates the allegations set forth in paragraphs 1 through 163 as if fully stated herein.

205. Ohio asserts this cause of action pursuant to its role as public trustee of natural resources and its inherent *parens patriae* authority to defend a quasi-sovereign interest, and does not here assert or usurp claims on behalf of any individual or non-State entity harmed in his or her person or property by Defendants' conduct.

206. Defendants failed to exercise ordinary care because a reasonably careful company that learned of its product's toxicity, harmfulness to humans, and harmfulness to the natural environment would not manufacture or distribute that product, or would warn of its toxic and environmentally hazardous properties, or would take steps to enhance the safety and/or reduce the toxicity and environmental persistence of the product.

207. Defendants failed to exercise ordinary care because a reasonably careful company would not continue to manufacture or distribute AFFF products

containing or breaking down into PFOS and/or PFOA in mass quantities and to the extent that Defendants manufactured and distributed them.

208. Defendants were grossly negligent because they failed to exercise even slight care, placing revenue and profit generation above human and environmental health and safety.

209. Defendants owed the State and its citizens a duty of care in the manufacture, distribution, marketing, promotion, and sale of AFFF products containing or breaking down into PFOS and/or PFOA because it was foreseeable to Defendants that their products, once used as intended, would end up in Ohio's public natural resources, including waterways, waterbodies, aquifers, soils, lands and submerged lands, sediments, fish and animal tissue, and biota.

210. Defendants' negligent conduct and the presence of PFOS and/or PFOA released from Defendants' products in Ohio caused and continue to cause injury to the physical and economic health and well-being of Ohio citizens.

211. Ohio has suffered and will continue to suffer damages to its public natural resources and public fisc as a result of Defendants' negligent conduct and the presence of PFOS and/or PFOA released from Defendants' products within the State.

212. Defendants are jointly and severally liable for all such damages, and the State is entitled to recover all such damages and other relief as set forth below.

FIFTH CAUSE OF ACTION
PUBLIC NUISANCE

213. Ohio realleges and incorporates the allegations set forth in paragraphs 1 through 163 as if fully stated herein.

214. Ohio asserts this cause of action pursuant to its role as public trustee of natural resources and its inherent *parens patriae* authority to defend a quasi-sovereign interest, and does not here assert or usurp claims on behalf of any individual or non-State entity harmed in his or her person or property by Defendants' conduct.

215. Defendants manufactured, distributed, marketed, promoted, and sold AFFF products containing or breaking down into PFOS and/or PFOA in a manner that created or participated in the creation of a public nuisance that is harmful to human and environmental health and obstructs the free use of public natural resources.

216. Defendants intentionally manufactured, distributed, marketed, promoted, and sold AFFF products containing or breaking down into PFOS and/or PFOA with the knowledge that they were causing and would continue to cause environmental contamination of Ohio's public natural resources, including waterways, waterbodies, aquifers, groundwater, soils, lands and submerged lands, sediments, fish and animal tissue, and biota

217. Defendants knew, or in the exercise of reasonable care should have known, that their products would end up in Ohio's public natural resources, including waterways, waterbodies, aquifers, groundwater, certain drinking water supplies, lands and submerged lands, soils, sediments, fish and animal tissue, and biota.

218. Defendants' conduct and the presence of PFOS and/or PFOA released from Defendants' products annoy, injure, and endanger the comfort, repose, health, and safety of others.

219. Defendants' conduct and the presence of PFOS and/or PFOA released from Defendants' products in Ohio public natural resources are injurious to human, animal, and environmental health.

220. An ordinary person would be reasonably annoyed or disturbed by the presence of toxic PFOS and/or PFOA that endanger the health of fish, animals, and humans, and degrade water quality and marine habitats as well as soils and sediments, and biota within Ohio.

221. The seriousness of the environmental and human health risk far outweighs any social utility of Defendants' conduct in manufacturing, distributing, marketing, promoting, and selling AFFF products containing or breaking down into PFOS and/or PFOA and concealing the dangers posed to human and environmental health.

222. The rights, interests, and inconvenience to Ohio and the general public far outweighs the rights, interests, and inconvenience to Defendants, who profited heavily from the manufacture, distribution, marketing, promotion, and sale of AFFF products containing or breaking down into PFOS and/or PFOA.

223. Defendants' conduct caused and continues to cause harm to the State and its citizens.

224. Ohio suffered and continues to suffer damage from Defendants' products, including costs to remove the PFOS and/or PFOA released from Defendants' products that have invaded Ohio public natural resources, prevent PFOS and/or PFOA released from Defendants' products from injuring additional Ohio public natural resources, and restore those public natural resources whose use has been lost. The injury to Ohio public natural resources is especially injurious to the State in its proprietary and public capacities, as well as its natural resource trustee capacity.

225. The State is incurring and will continue to incur costs to investigate, monitor, analyze, and remediate PFOS and/or PFOA contamination caused by Defendants' products in Ohio public natural resources.

226. Defendants knew, or in the exercise of reasonable care should have known, that the manufacture, distribution, marketing, promotion, and sale of AFFF

products containing or breaking down into PFOS and/or PFOA was causing and would cause the type of contamination now found in Ohio public natural resources.

227. Defendants knew, or in the exercise of reasonable care should have known, that PFOS and/or PFOA would contaminate waterbodies, including potentially drinking water supplies, degrade marine habitats and endanger fish, birds, and animals, and contaminate soils, sediments, and biota within Ohio.

228. In addition, Defendants knew or should have known that PFOS and/or PFOA are associated with serious illnesses, including high cholesterol, thyroid disease, adverse reproductive effects, pregnancy-induced hypertension, preeclampsia, as well as testicular and kidney cancer, and that humans may be exposed to PFOS and/or PFOA including through ingestion of contaminated fish or water, among other things.

229. It was foreseeable to Defendants that humans may be exposed to PFOS and/or PFOA through, *e.g.*, drinking contaminated water or eating fish from contaminated areas.

230. Accordingly, Defendants had a duty to cease manufacturing, distributing, marketing, promoting, and selling AFFF products containing or breaking down into PFOS and/or PFOA, or to reformulate such products as alleged above, but failed to do so.

231. Defendants also had a duty to warn about the dangers of AFFF products containing or breaking down into PFOS and/or PFOA but failed to do so, as alleged above.

232. As a direct and proximate result of Defendants' creation of a public nuisance, Ohio has suffered and continues to suffer monetary losses, including loss of value and loss of use of Ohio public natural resources, in amounts to be proven at trial.

233. Defendants are jointly and severally liable for all such damages, and the State is entitled to recover all such damages and other relief as set forth below.

SIXTH CAUSE OF ACTION
TRESPASS

234. Ohio realleges and incorporates the allegations set forth in paragraphs 1 through 163 as if fully stated herein.

235. Ohio asserts this cause of action pursuant to its role as public trustee of natural resources and its inherent *parens patriae* authority to defend a quasi-sovereign interest, and does not here assert or usurp claims on behalf of any individual or non-State entity harmed in his or her person or property by Defendants' conduct.

236. Defendants' conduct wrongfully contaminated and caused injury to Ohio public natural resources.

237. Defendants acted intentionally and unreasonably while knowing, or having reason to know, that the State did not give Defendants authorization to act in a manner that would contaminate and cause injury to Ohio public natural resources.

238. Due to Defendants' wrongful and intentional conduct in introducing AFFF products containing or breaking down into PFOS and/or PFOA into Ohio, which Defendants knew would contaminate and cause injury to the public natural resources of the State, Ohio suffered and will continue to suffer damages.

239. Defendants' wrongful and intentional conduct in introducing AFFF products containing or breaking down into PFOS and/or PFOA into the State, which Defendants knew would contaminate and cause injury to the public natural resources of the State, was and is the direct factual and legal cause of the injury to Ohio.

240. Defendants are jointly and severally liable for all such damages, and the State is entitled to recover all such damages and other relief as set forth below.

SEVENTH CAUSE OF ACTION
UNJUST ENRICHMENT

241. Ohio realleges and incorporates the allegations set forth in paragraphs 1 through 163 as if fully stated herein.

242. Ohio asserts this cause of action on its own behalf.

243. Ohio has incurred and will continue to incur expenses in connection with PFOS and/or PFOA contamination within the State caused by use of Defendants' AFFF products containing or breaking down into PFOS and/or PFOA, including investigative, assessment, and remediation costs.

244. Defendants are responsible for the PFOS and/or PFOA contamination within the State caused by use of their AFFF products containing or breaking down into PFOS and/or PFOA that Ohio has addressed and will address, and in fairness, Defendants should have paid these costs. It would be unjust for Defendants to retain the benefit of Ohio's expenditures in connection with the PFOS and/or PFOA contamination they caused within the State.

245. Ohio requests an injunction ordering Defendants to return all monies by which Defendants were unjustly enriched as a result of Ohio's expenditures in connection with the PFOS and/or PFOA contamination they caused within the State.

246. Defendants are jointly and severally liable for all such damages, and the State is entitled to recover all such damages and other relief as set forth below.

PRAYER FOR RELIEF

Ohio prays for judgment against Defendants, jointly and severally, as follows:

A. Compensatory damages, in excess of $25,000, to Ohio according to proof;

B. Damages for injury to Ohio public natural resources, including the economic impact to the State and its residents and costs to assess, investigate, monitor, analyze, and remove the PFOS and/or PFOA contamination that was introduced into the State through Defendants' AFFF products, to prevent Defendants from injuring additional public natural resources, and to restore public natural resources whose use has been lost or impaired, on a statewide basis;

C. Any other damages, including punitive or exemplary damages, as permitted by law;

D. Award of present and future costs to clean up the PFOS and/or PFOA contamination complained of herein;

E. An injunction ordering Defendants to return all monies by which Defendants were unjustly enriched as a result of the State's expenditures in connection with PFOS and/or PFOA contamination within the State caused by use of Defendants AFFF products containing or breaking down into PFOS and/or PFOA;

F. Litigation costs and attorneys' fees as permitted by law;

G. Pre-judgment and post-judgment interest on all monies awarded, as permitted by law;

H. Such other and further relief as the Court deems just and proper.

DATED: December 21, 2018

MICHAEL DeWINE
ATTORNEY GENERAL OF OHIO

By: */s/ John F. Bodie, Jr.*

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Special Counsel for Plaintiff the State of Ohio

JURY DEMAND

Ohio respectfully requests trial by jury on all claims so triable.

/s/ John F. Bodie, Jr.

Exhibit 1

FC in Blood (Alma)

c: Rick Davis
10/24/77
T.O.

Interoffice Correspondence 3M

Subject:

October 19, 1977

CONFIDENTIAL

RECEIVED
OCT 20 1977
L. J. SCHEUERMAN

TO: J. D. LAZERTE
T. J. SCHEUERMAN
F. A. UBEL

FROM: L. C. KROGH

In order to help you with your preparations for the presentation on November 7 to the Corporate Responsibility Committee, I am enclosing copies of the transparencies used in the last report.

I was in error yesterday, we last reported to the Corporate Responsibility Committee on November 8, 1977, not in February.

LCK:jmb
encl.

3MA10067215

Exhibit
1145
State of Minnesota v. 3M Co.,
Court File No. 27-CV-10-28862

Trial Exhibit 1202

3M_MN00000479

ORGANIC FLUORINE COMPOUNDS IN BLOOD

CHRONOLOGY

AUGUST 22, 1975	- DR. J. D. LA ZERTE RECEIVES CALL FROM W. S. GUY OF UNIVERSITY OF ROCHESTER.
AUGUST 25, 1975	- W. S. GUY, D. R. TAVES, AND W. S. BREY, JR. PRESENT A PAPER AT CHICAGO ACS MEETING ENTITLED "CHARACTERISTICS AND CONCENTRATIONS OF ORGANIC FLUORO COMPOUNDS FOUND IN HUMAN TISSUES."
SEPTEMBER 17-21, 1975	- CENTRAL RESEARCH ANALYTICAL TO COMPARE $C_7F_{15}COOH$ AND $C_8F_{17}SO_3H$ NMR SPECTRA WITH THAT REPORTED BY GUY ET AL.
SEPTEMBER 22, 1975	- TAVES CALLS J. D. LA ZERTE TO DETERMINE IF 3M WILL FURTHER ANALYZE THEIR SAMPLE OF FLUOROCHEMICAL. ALSO ASKS 3M TO OPEN CONTENTS OF FDA PETITION ON "SCOTCHBAN."
OCTOBER, 1975	- CENTRAL RESEARCH ANALYTICAL AGREES TO DETERMINE QUANTITY AND CHARACTER OF ORGANIC FLUORINE COMPOUNDS IN HUMAN BLOOD.
NOVEMBER 6, 1975	- CENTRAL RESEARCH REPORTS THAT $C_8F_{17}SO_3H$ SPECTRA MATCHES THAT PRESENTED BY GUY, ET AL.
DECEMBER 16, 1975	- LA ZERTE, FREIER, AND LONG OF 3M VISIT GUY AND TAVES AT THE UNIVERSITY OF ROCHESTER. 3M PROPOSES, AND GUY AND TAVES AGREE THAT 3M WILL ATTEMPT TO ISOLATE AND IDENTIFY ORGANIC FLUORINE COMPOUNDS IN HUMAN BLOOD.

3MA10067216

3M_MN00000480

CHRONOLOGY - PAGE 2

FEBRUARY 17, 1976	- CENTRAL RESEARCH ANALYTICAL DEVELOPS AN ACCURATE ANALYTICAL METHOD FOR DETERMINING PARTS PER BILLION QUANTITIES OF ORGANIC FLUORINE COMPOUNDS IN HUMAN BLOOD. METHOD TESTED ON BLOOD FROM AMERICAN RED CROSS AND VALUE AGREES WITH THOSE IN LITERATURE.
APRIL 14, 1976	- FOUR LABORATORY PERSONNEL HAVE BLOOD SAMPLES ANALYZED. CONCENTRATION OF ORGANIC FLUORINE COMPOUNDS IN SOME PERSONNEL 100 TIMES NORMAL.
JUNE 29, 1976	- SOME CHEMOLITE PERSONNEL SHOW ORGANIC FLUORINE COMPOUNDS AT 1,000 TIMES NORMAL.
AUGUST 23, 1976	- CORDOVA PERSONNEL EXPOSED TO FLUORO-CHEMICALS HAVE UP TO 50 TIMES NORMAL VALUES.
AUGUST 26, 1976	- CENTRAL RESEARCH ISOLATES AND IDENTIFIES ORGANIC FLUORINE COMPOUNDS FROM BLOOD OF CHEMOLITE PERSON AS $C_7F_{15}COOH$.
SEPTEMBER 9, 1976	- MICE FED "SCOTCHBAN." HAD 4,000 TIMES NORMAL ORGANIC FLUORINE COMPOUNDS.
SEPTEMBER 17, 1976	- $C_8F_{17}SO_3H$ IDENTIFIED AS ORGANIC FLUORINE COMPOUND IN MICE FED "SCOTCHBAN."
OCTOBER 8, 1976	- DECATUR PLANT PERSONNEL FOUND TO HAVE UP TO 300 TIMES NORMAL LEVELS.
	- INDIVIDUALS EXPOSED TO FLUOROCHEMICALS OVER 20 YEARS AGO HAVE NORMAL ORGANIC FLUORINE COMPOUND LEVELS.

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3M_MN00000481

CHRONOLOGY - PAGE 3

OCTOBER 28, 1976

- DR. LEON SINGER OF THE BIOCHEMISTRY DEPARTMENT, UNIVERSITY OF MINNESOTA, CALLS TO OBTAIN SAMPLES OF $C_7F_{15}COOH$.
- DR. SINGER REPORTS THAT HE HAS HAD CONVERSATIONS WITH TAVES. ALSO REPORTS THAT ANIMALS FED INORGANIC FLUORIDE SHOW INCREASES IN ORGANIC FLUORINE BLOOD LEVEL.

3MA10067218

3M_MN00000482

ORGANIC FLUORINE COMPOUNDS IN BLOOD

STATUS AT 3M

1. NO EVIDENCE NOW OF RELATED HEALTH PROBLEMS.
2. 3M MEDICAL DEPARTMENT INITIATING PROGRAM TO STUDY BLOOD CHEMISTRY OF EXPOSED PERSONNEL.
3. NO EVIDENCE THAT THE PROBLEM EXISTS WITH 3M'S CUSTOMERS.
4. FUNDS ARE BUDGETED TO CONTINUE THE PROGRAM STUDY UNTIL WE ARE SATISFIED:
   - (A.) THAT THERE IS NO HEALTH HAZARD INCURRED BY 3M EMPLOYEES; AND,
   - (B.) THAT THE CONTINUED SALE OF OUR FLUOROCHEMICALS FOR VARIOUS PURPOSES DOES NOT ENDANGER THE PUBLIC'S HEALTH.

3MA10067219

3M_MN00000483

FLUORINE CONTENT OF BLOOD SERUM

	Organic Fluorine P.P.B.	Inorganic Fluorine P.P.B.
1. LITERATURE VALUES	2 - 130 Ave. 30	3 - 170 Ave. 20
2. 3M CONTROLS	10 - 80	40 - 60
3. LABORATORY BUILDING 236	430 - 3,100	30 - 360
4. CORDOVA	160 - 630	50 - 870
5. CHEMOLITE	510 - 38,800	50 - 90
6. DECATUR	130 - 9,840	40 - 210

3MA10067220

3M_MN00000484

SOURCES OF FLUORINE

INORGANIC		
	SnF_2	TOOTHPASTE, DENTAL CARE.
	F^-	FLUORIDIZED WATER
ORGANIC		
	3M	$CF_3CF_2CF_2CF_2CF_2CF_2CF_2COOH$ $CF_3CF_2CF_2CF_2CF_2CF_2CF_2CF_2SO_3H$
OTHERS		STEROIDS TRANQUILIZERS ANESTHETICS HERBICIDES TEFLON AEROSOL PROPELLANTS REFRIGERANTS

3MA10067221

3M_MN00000485

Exhibit 2

CENTRAL ANALYTICAL LABORATORY

Report No. 6967

Date August 4, 1978

Subject: IRDC 137-092: FC-95/Monkey

Requestor: J.E. Long Dept. Name Toxicology Proj. No. 9172110004

Request No. A69507 Dated 8/3/78

Report:

Reference 137-087 by IRDC was a 90 day subacute Rhesus monkey toxicity study of FC-95. Incorrect (too high) feeding levels were used and all animals died within the first few days. The feeding levels were lowered and the study started again as 137-092. The serum and livers were each individually submitted for analysis. Each group contained 4 monkeys, 2 male and 2 female as follows:

Group	Dosage Level	Survival
I	0	4/4
II	0.5 mg/kg/day	4/4
III	1.5 mg/kg/day	4/4
IV	4.5 mg/kg/day	0/4

SERUM ANALYSIS

Monkey	Dosage Level	FC-95 in Serum (ppm)①
Blank on Method	-	2
7355M	0	40
7358M	0	20
7368F	0	15
7463M	0.5 mg/kg/day	150
7466F	"	150
7462M	1.5 mg/kg/day	250
7500F	"	275

① Our newly developed pyrolysis method was used. Precision is estimated to be ±10 - 25%.

Exhibit 1181
State of Minnesota v. 3M Co., Court File No. 27-CV-10-28862

3M_MN02343995

The high levels of FC-95 in the control serum cannot be explained. For example, 7355M was run on 2 different days with the same experimental results. Perhaps a total fluorine should be run on this sample to determine if this level of F is present. See Table below containing liver results for further discussion (Footnote 2).

LIVER ANALYSIS

Monkey	Dosage Level	FC-95 in Liver(mg)①	FC-95 in Liver(ppm)
7355M	0	3000	50
7368F	0	1500②	20②
7463M	0.5 mg/kg/day	7000	100
7466F	"	8000	100
7462M	1.5 mg/kg/day	45000	650
7500F	"	40000	600
7484M	4.5 mg/kg/day	40000	650
7502F	"	80000	1000

① The livers were stored in the refrigerator several months prior to analysis resulting in the separation of some liquid. The high values and physical size necessitates taking only partial livers in which case the same ratio (estimated visually) of solid to separated liquid was taken for analysis. In addition, recovery of FC-95 was estimated from experiments where FC-95 was added to a control liver.

② Because of a higher than expected value for the control, a 0.133 g liver sample was analyzed for total fluorine. The fluorine value calculated as FC-95 is equivalent to 11 ppm FC-95 in the liver. As time permits, more work should be done to verify these "high" control levels.

Jon Belisle

cc:R.A. Prokop 236-3

Exhibit 3

AR226-0447

International Research and Development Corporation

SPONSOR: 3M Company

COMPOUND: Fluorad® Fluorochemical FC-143

SUBJECT: Ninety Day Subacute Rhesus Monkey Toxicity Study.

Edwin I. Goldenthal, Ph.D.
Vice President and
Director of Research

Collaborators:

D. C. Jessup, Ph.D., Associate Director of Research
R. G. Geil, D.V.M., Vice President and Director of Pathology
J. S. Mehring, Ph.D., Director of Large Animal Toxicology

Date: November 10, 1978

137-090

TABLE OF CONTENTS
(Continued)

I. SYNOPSIS

In a ninety day oral study in rhesus monkeys, Fluorad® Fluorochemical FC-143 was administered at dosage levels of 0 (control, treated only with 0.5% Methocel®), 3, 10, 30 and 100 mg/kg/day. Two male and two female monkeys were initiated at each dosage level and also in a control group. The monkeys were observed twice daily for general physical appearance and behavior and pharmacotoxic signs. Body weights were recorded weekly. Hematological, biochemical and urinalysis studies were conducted once in the control period, at the end of the first and third months of study.

The monkeys treated with the higher dose, (100 mg/kg/day) all died during weeks 2 through 5 of the study. At the 30 mg/kg/day dosage level, three monkeys died during weeks 7-12. They all showed signs of toxicity in the gastrointestinal tract (anorexia, emesis, sometimes brown in color, black stools), pale face and gums, swollen face and eyes, slight to severe decreased activity and prostration. The monkeys of the 30 and 100 mg/kg/day dosage level showed body weight losses from the first week of the study.

Because of the early deaths of the monkeys at the 100 mg/kg/day dosage level, the clinical laboratory tests were not conducted.

The monkeys at the 30 mg/kg/day dosage level showed, in the first month of the study, slight increase in prothrombin time and in activated partial thromboplastin time (A.P.T.T.) values, as well as decreased alkaline phosphatase activity in the serum (statistically significant). Only one monkey from this dosage level in this period showed a low albumin value. At the end of the study, the only remaining monkey from the 30 mg/kg/day dosage level showed apparent anemia, low blood glucose, alkaline phosphatase, total protein and albumin values.

There was no mortality at the 10 mg/kg/day dosage level. One monkey had black stool on several days in week 12 and occasionally

anorexia and one monkey exhibited pale face and gums. At this dosage level there was a very slight increase in the activated P.T.T. values in the female monkeys during the first month of the study (not statistically significant). There were no changes in the other indices and no changes in the body weight. In single monkeys from the 3 and 10 mg/kg/day dosage levels, there were trends toward decreased alkaline phosphatase in the serum.

In the control and the 3 mg/kg/day dosage level there was no mortality, no changes in the body weights and no signs of toxicity. Soft stool, diarrhea or emesis were observed occasionally.

The mortality and the above mentioned signs of toxicity in the 30 and 100 mg/kg/day dosage levels were compound-related. There was a trend toward the same signs of toxicity in single monkeys at the 10 mg/kg/day dosage level. The 3 mg/kg/day dosage level seems to be free of signs of toxicity. There is an evident relationship between the administered doses and the degree of the toxicity.

No gross or microscopic lesions which were considered compound-related were seen in tissues other than the adrenals, bone marrow, spleen and lymph nodes for male and female monkeys at the 30 and 100 mg/kg/day dosage levels. Microscopically, the adrenals from male and female monkeys at the 30 and 100 mg/kg/day dosage levels had compound-related marked diffuse lipid depletion; the bone marrow from male and female monkeys at the 30 and 100 mg/kg/day dosage levels had compound-related slight to moderate hypocellularity; the spleen and lymph nodes from male and female monkeys at the 30 and 100 mg/kg/day dosage levels had compound related moderate atrophy of lymphoid follicles.

Statistically significant variations in sex group mean weights of a few organs occurred between the control and experimental groups. These variations were of unknown biological significance and were not accompanied by morphologic alterations.

II. COMPOUND

The compound was received from 3M Company, Saint Paul, Minnesota on October 24, 1977 as shown below:

Label	Description
Fluorad® Fluorochemical FC-143 3M Stock No. 98-0211-0008-0 Lot 340	white powder

III. CLINICAL STUDIES

A. METHODS:

1. General Procedure:

Ten male rhesus monkeys (weighing from 2.60 to 3.90 kilograms) and 10 females (weighing from 2.95 to 3.80 kilograms) were initiated on this study. The monkeys were purchased from Primate Imports Corporation, Port Washington, N. Y. 11050. The monkeys were housed individually in hanging wire mesh, "squeeze type" cages and maintained in a temperature, humidity and light controlled enviroment. Purina® Monkey Chow® was fed twice each day and fresh apples were fed 3 times a week. Water was available ad libitum.

During the conditioning period, the monkeys were tattooed on the inner surface of the thigh and intrapalpebral tuberculin tests were conducted. Tuberculin tests were conducted at bimonthly intervals during the treatment period. Also a complete physical examination was conducted by the staff veterinarian prior to initiation of compound administration. Only monkeys in good health were selected for the study.

This study was initiated on January 11, 1978. Terminal sacrifices were conducted on April 12, 1978.

2. Compound Administration:

At the end of the conditioning period the monkeys were divided into five groups on a random basis, so that the initial average body weights were similar:

Number of Monkeys		Dosage Level
Male	Female	
2	2	Control
2	2	3 mg/kg/day
2	2	10 mg/kg/day
2	2	30 mg/kg/day
2	2	100 mg/kg/day

The test compound, suspended in 0.5% Methocel®, was administered by gavage, 7 days each week. All doses were given in a constant volume. Also the same volume of 0.5% Methocel® was given to the vehicle control group. Individual daily doses were based upon the body weights obtained weekly.

3. Observations:

The monkeys were observed twice daily for general physical appearance and behavior and pharmacotoxic signs. Individual body weights were recorded weekly. General physical examinations were conducted in the control period and monthly during the study.

4. Clinical Laboratory Tests:

Blood and urine samples were obtained for analysis from all monkeys once during the control period and at 1 and 3 months of study. The monkeys were fasted overnight prior to the collection of blood and urine samples.

a. Hematology:

Hematological studies included: hemoglobin[1], hematocrit[2], erythrocyte count[3], total[3] and differential leucocyte counts, reticulocyte count[4], platelet count[5], prothrombin time[6], activated partial thromboplastin time[7] (A.P.T.T.). Mean corpuscular hemoglobin, mean corpuscular volume and mean corpuscular hemoglobin concentration were calculated.

b. Biochemistry:

Biochemical studies included: fasting blood glucose[8], blood urea nitrogen[8], serum alkaline phosphatase[8], serum glutamic oxalacetic and pyruvic transaminase activities[8], cholesterol[9], total protein[9], albumin[8], sodium[10], potassium[10], chloride[9], inorganic phosphate[9], γ-glutamyl transpeptidase[11] (γ-G.T.P.) and creatinine phosphokinase[9].

c. Urinalysis:

Urinalysis included: measurement of volume, pH[12] and specific gravity; description of color and appearance; qualitative tests for protein[12], glucose[12], ketones[12], occult blood[12] and microscopic examination of the sediment.

d. Statistical Analysis:

Analysis of body weights and clinical laboratory tests were performed. All statistical analyses compared the treatment groups with the control group, by sex. The tests were compared by analysis of variance (one-way classification) Bartlett's test for homogeneity and the appropriate t-test (for equal or unequal variances) as described by Steel and Torrie[13] using Dunnett's[14] multiple comparison tables to judge significance of differences.

B. RESULTS:

1. General Behavior, Appearance and Survival:

There was no mortality in monkeys at 0, 3 and 10 mg/kg/day dosage levels.

The monkeys from the control and 3 mg/kg/day dosage levels did not show any unusual behavior or signs of toxicity. Soft stool or moderate to marked diarrhea were noted occasionally. Frothy emesis was also noted occasionally.

At the 10 mg/kg/day dosage level the monkeys did not show any unusual signs of toxicity, except Monkey 7363. In week 7 its face appeared swollen and pale. It had been occasionally anorexic in week 4 and black stools appeared for several days in week 12 of the study.

At the 30 mg/kg/day dosage level, three monkeys died during weeks 7, 12 and 13 of the study. From week 4, the monkeys were anorexic. Slight to moderate and sometimes severe decreased activity was noted occasionally to frequently for the four monkeys. Emesis and ataxia were very rarely noted, for one monkey.

Swollen face, eyes and vulva, as well as pallor of the face and gums were noted. From week 6, for two monkeys, black stools were noted. Monkey 7387 showed slight to moderate dehydration and ptosis of the eyelids.

All monkeys from the 100 mg/kg/day dosage level died during weeks 2 through 5 of study. They showed the same symptoms of toxicity as the previous group, but they appeared sooner in the study (from week 1) and were more marked: anorexia, frothy emesis (sometimes brown in color) pale face and gums, swollen face and eyes, decreased activity from slight to severe, prostration and body trembling.

2. Body Weights (Tables 1-3):

Changes in body weight were similar for monkeys from the control and the 3 and 10 mg/kg/day dosage levels. Monkeys at the 30 and 100 mg/kg/day dosage levels lost body weight after the first week of study. There was statistically significant decreases in the body weight for the male monkeys at the 30 mg/kg/day dosage level in week 13 of the study. The female monkeys of the same dosage level and the monkeys from the 100 mg/kg/day dosage level were dead in this period.

3. Laboratory Test (Tables 4-15):

a. Hematology:

There were no noteworthy changes in monkeys from the 3 and 10 mg/kg/day dosage levels. In the first month of the study there was a slight increase (not statistically significant) of the A.P.T.T. values in the females at the 10 mg/kg/day dosage level and a statistically significant increase of the A.P.T.T. and prothrombin time values in monkeys at the 30 mg/kg/day dosage level. In the third month of the study there was a high increase in the above mentioned indices for the one surviving monkey from the 30 mg/kg/day dosage level. The same monkey (#7455) had pronounced anemia as well.

The statistically significant increase in the hematocrit in monkeys at the 10 mg/kg/day dosage level and in the platelet count in monkeys at the 3 mg/kg/day dosage level at 3 months of study, were within the normal physiological limits.

b. Biochemistry:

There were no noteworthy changes in monkeys from the control, 3 and 10 mg/kg/day dosage level. Only one monkey from the 3 mg/kg/day dosage level and one monkey from the 10 mg/kg/day dosage level showed trends toward decreases of alkaline phosphatase (432 and 474 units/l, respectively), without statistical significance.

In the first month of the study, decrease in serum alkaline phosphatase was noted in monkeys at the 30 mg/kg/day dosage level (statistically significant) and in one monkey in the same dosage level, the albumin in the serum was lower (3.22 g/100ml). The one surviving monkey (7455) from the 30 mg/kg/day dosage level showed decreasing of: blood sugar (66 mg/100ml), total protein (5.52 g/100ml) with albumin (2 g/100ml) and alkaline phosphatase (360 units/l) and slightly elevated cholesterol (240 mg/100ml).

c. Urinalysis:

No changes considered to be related to compound were seen in the urinalysis studies.

IV. PATHOLOGICAL STUDIES

A. METHODS:

1. Gross Pathology:

After completion of the compound administration period all surviving monkeys were anesthetized with Sernylan®*, exsanguinated and necropsied. At necropsy, the heart, liver, adrenals, spleen, pituitary, kidneys, testes/ovaries and brain were weighed and representative tissues were collected in buffered neutral 10% formalin. Eyes were fixed in Russell's fixative. The thyroid/parathyroid was weighed after fixation.

Monkeys which died during the study were necropsied as above.

2. Histopathology:

Microscopic examination of formalin fixed hematoxylin and eosin stained paraffin sections was performed for all monkeys in the control and treatment groups. The following tissues were examined:

adrenals	kidneys	lumbar spinal cord
aorta	liver	pituitary
bone	lung	stomach
brain	skin	testes/ovaries
esophagus	mesenteric lymph node	thyroid
eyes	retropharyngeal lymph node	parathyroid
gallbladder	mammary gland	thymus
heart (with coronary vessels)	nerve (with muscle)	trachea
duodenum	spleen	tonsil
ileum	pancreas	tongue
jejunum	prostate/uterus	urinary bladder
cecum	rib junction (bone marrow)	vagina
colon	salivary gland	tattoo
rectum		

and any other tissue(s) with lesions

*Phencyclidine HCl - Bio-Ceutic Laboratories, Inc., St. Joseph, Missouri.

B. RESULTS:

1. Gross Pathology (Table 16) and Organ Weights (Table 17):

No gross lesions considered compound related were seen in male and female rhesus monkeys which died on study or were sacrificed after 90 days of study.

Statistically significant variations in sex group mean weights of few organs occurred between the control and experimental groups. The following statistically significant organ weight variations occurred:

Organ	Dosage Level mg/kg/day	Sex	Weight	Change	P<
Heart	10	F	absolute,relative	decrease,decrease	0.05,0.01
Brain	10	F	absolute	decrease	0.01
Pituitary	3	M	relative	increase	0.05

The biological significance of these variations is unknown. These organ weight variations were not accompanied by morphologic changes which were considered compound related.

2. Histopathology (Table 18):

One male and two female rhesus monkeys at the 30 mg/kg/day dosage level and all male and female rhesus monkeys at the 100 mg/kg/day dosage level had marked diffuse lipid depletion in the adrenals. All male and female rhesus monkeys at the 30 and 100 mg/kg/day dosage levels had slight to moderate hypocellularity of the bone marrow. All male and female rhesus monkeys at the 30 and 100 mg/kg/day dosage levels had moderate atrophy of lymphoid follicles in the spleen. One female at the 30 mg/kg/day dosage level and all male and female rhesus monkeys at the 100 mg/kg/day dosage level had moderate atrophy of the lymphoid follicles in the lymph nodes.

No microscopic changes considered compound related were seen in the adrenals, bone marrow, spleen and lymph nodes of male and female rhesus monkeys at the 3 and 10 mg/kg/day dosage levels. No microscopic

lesions in tissues other than the adrenals, bone marrow, spleen and lymph nodes at the 30 and 100 mg/kg/day dosage levels were considered compound-related.

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FC-143: Ninety Day Subacute Rhesus Monkey Toxicity Study.

TABLE 1. Mean Body Weights of Monkeys Week 13 of Study.

Sex	Group I (Control)	Group II (3 mg/kg/day)	Group III (10 mg/kg/day)	Group IV (30 mg/kg/day)	Group V (100 mg/kg/day)
M	3.78	3.50	3.68	2.30*	dead
F	3.55	3.68	3.78	dead	dead

*Statistical significance.

FC-143: Ninety Day Subacute Rhesus Monkey Toxicity Study.

TABLE 2. Individual Body Weights, Kilograms.

Group, Monkey Number	Sex	Control 1	Control 2	Week of Study 1	2	3	4	5	6	7	8	9	10	11	12	13
Control:																
7362	M	3.15	3.30	3.15	3.30	3.35	3.10	3.20	3.20	3.00	3.15	3.20	3.05	3.20	3.40	3.50
7365	M	3.50	3.50	3.50	3.50	3.50	3.40	3.55	3.60	3.60	3.80	3.75	3.75	3.80	4.00	4.05
7336	F	3.05	3.20	3.25	3.25	3.35	3.15	3.00	3.15	3.20	3.30	3.45	3.30	3.35	3.35	3.60
7386	F	3.90	3.70	3.70	3.65	3.55	3.45	3.40	3.55	3.40	3.40	3.55	3.40	3.50	3.50	3.50
Mean		3.40	3.43	3.40	3.43	3.44	3.28	3.29	3.38	3.30	3.41	3.49	3.38	3.46	3.56	3.66
3 mg/kg/day:																
7364	M	3.70	3.90	3.85	3.95	3.85	3.85	3.80	3.80	3.85	4.10	4.10	4.05	4.05	4.20	4.30
7366	M	2.60	2.60	2.70	2.60	2.65	2.65	2.70	2.70	2.50	2.70	2.70	2.45	2.55	2.50	2.70
7384	F	3.55	3.60	3.70	3.80	3.80	3.80	3.70	3.70	3.60	3.55	3.80	3.55	3.70	3.90	3.75
7385	F	3.50	3.55	3.45	3.45	3.45	3.45	3.40	3.40	3.50	3.55	3.60	3.40	3.30	3.40	3.60
Mean		3.34	3.41	3.43	3.45	3.44	3.44	3.40	3.40	3.36	3.48	3.55	3.36	3.40	3.50	3.59
10 mg/kg/day:																
7363	M	3.55	3.70	3.70	3.65	3.65	3.65	3.65	3.60	3.60	3.70	3.65	3.75	3.85	3.90	3.90
7458	M	3.10	3.10	3.25	3.20	3.10	3.05	2.95	3.20	3.00	3.15	3.10	3.10	3.25	3.25	3.45
7328	F	3.30	3.30	3.45	3.40	3.40	3.30	3.20	3.30	3.25	3.45	3.60	3.50	3.40	3.60	3.75
7383	F	3.60	3.60	3.50	3.80	3.60	3.55	3.50	3.60	3.60	3.65	3.80	3.65	3.75	3.75	3.80
Mean		3.39	3.43	3.48	3.51	3.44	3.39	3.33	3.43	3.36	3.49	3.54	3.50	3.56	3.63	3.73

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TABLE 2. Cont.

Individual Body Weights, Kilograms.

Group, Monkey Number	Sex	Control 1	Control 2	Week of Study 1	2	3	4	5	6	7	8	9	10	11	12	13
30 mg/kg/day:																
7367	M	3.40	3.40	3.25	3.25	3.10	2.95	2.65	2.30	2.10*	Died					
7455	M	3.50	3.30	3.20	3.05	2.85	2.65	2.45	2.50	2.55	2.60	2.70	2.70	2.65	2.50	2.30
7382	F	3.25	3.30	3.20	3.20	3.05	3.00	2.85	2.80	2.80	2.80	2.80	2.80	2.80	2.60	2.25* Died
7387	F	3.70	3.75	3.50	3.55	3.50	3.45	3.10	2.95	2.85	2.85	2.70	2.65	2.50	2.25*	Died
Mean		3.46	3.44	3.29	3.26	3.13	3.01	2.76	2.64	2.73	2.75	2.73	2.72	2.65	2.55	2.30
100 mg/kg/day:																
7361	M	3.50	3.85	3.50	3.30	3.00	2.55	2.40*	Died							
7456	M	3.10	3.10	2.60	2.70*	Died										
7335	F	2.80	2.95	2.70	2.45	2.05*	Died									
7381	F	3.85	3.80	3.55	3.20	2.80	2.60*	Died								
Mean		3.31	3.43	3.09	2.98	2.90	2.55									

*Terminal weight not included in mean.

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TABLE 3. T-Test Comparison of Body Weights.

Study Week	Sex	Control	3 mg/kg/day	10 mg/kg/day	30 mg/kg/day	100 mg/kg/day
13	M	3.78	3.50	3.68	2.30[a]	-
	F	3.55	3.68	3.78	-	-

*p<0.05
**p<0.01
[a]Not included in statistical analysis due to only one surviving animal.
- Line indicates animals had died prior to week 13.

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TABLE 4. Means and Significance of Hematological Values.

Hematology	Month of Study	Control	3 mg/kg/day	10 mg/kg/day	30 mg/kg/day
Erythrocytes, 10^6/cmm	1	4.46	4.26	4.71	4.53
	3	4.90	4.74	5.47	3.84[a]
Hemoglobin, g/100 ml	1	11.7	11.4	12.1	11.7
	3	12.9	12.7	13.3	9.7[a]
Hematocrit, %	1	38	37	39	36
	3	37	37	40**	30[a]
Platelets, 10^3/cmm	1	253	233	210	219
	3	210	285*	216	261[a]
Reticulocytes, %	1	0.2	0.5	0.5	0.2
	3	0.3	0.2	0.2	0.2[a]
Prothrombin Time, sec	1	12	12	13	15**
	3	11	11	11	30[a]
Activated P.T.T., sec	1	28	28	31	35**
	3	26	26	24	65[a]
Leucocytes, 10^3/cmm	1	9.49	9.78	9.93	8.44
	3	9.40	9.83	11.96	10.14[a]
Neutrophils, %	1	24	19	26	15
	3	16	19	25	36[a]
Lymphocytes, %	1	75	76	72	85
	3	80	76	67	54[a]
Eosinophils, %	1	1	5*	2	0
	3	3	3	6	3[a]
Monocytes, %	1	0	0	0	0
	3	1	2	2	7[a]
Basophils, %	1	0	0	0	0
	3	0	0	0	0[a]
MCV, μ^3	1	86	86	82	80
	3	75	78	73	78[a]
MCH, µµg	1	27	27	26	26
	3	26	27	24	25[a]
MCHC, g/100 ml	1	31	31	32	32*
	3	36	35	34	32[a]

*Significantly different from control group, $p<0.05$.
**Significantly different from control group, $p<0.01$.
[a]Value not used in statistical analysis due to only one animal surviving.

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TABLE 5.

Individual Hematological Values - Control 1.

Group, Monkey Number	Sex	Erythrocytes 10^6/cmm	Hemoglobin g/100 ml	Hematocrit %	Platelets 10^3/cmm	Reticulocytes %	Prothrombin Time sec	Activated P.T.T. sec	Leucocytes 10^3/cmm	Neutrophils Seg. %	Neutrophils Non-Seg. %	Lymphocytes[a] %	Eosinophils[a] %	Monocytes[a] %	Basophils[a] %	MCV μ^3	MCH µµg	MCHC g/100 ml
Control:																		
7362	M	5.08	13.0	40	207	0.1	13	29	10.96	36	1	62	1	0	0	79	26	33
7365	M	4.72	11.9	38	319	0.3	13	10	14.79	27	0	72	1	0	0	81	25	31
7336	F	5.27	12.8	39	226	0.6	14	29	7.86	38	0	59	3	0	0	74	24	33
7386	F	4.20	11.1	34	227	0.5	14	21	12.09	59	0	39	1	1	0	81	26	33
Mean		4.82	12.2	38	245	0.4	14	27	11.43	40	0	58	2	0	0	79	25	33
1 mg/kg/day:																		
7364	M	4.50	11.5	37	155	0.4	13	25	8.98	42	0	57	0	1	0	82	26	31
7366	M	4.48	12.0	37	297	0.3	14	29	7.39	41	0	59	0	0	0	83	27	32
7384	F	4.55	11.7	38	160	0.2	13	30	14.72	31	0	64	5	0	0	84	26	31
7385	F	4.19	11.4	35	145	0.6	13	24	8.16	38	0	59	3	0	0	84	27	33
Mean		4.43	11.7	37	232	0.4	13	27	9.81	38	0	60	2	0	0	83	27	32
10 mg/kg/day:																		
7363	M	5.24	13.7	42	264	0.4	13	31	12.97	46	0	49	5	0	0	80	26	33
7458	M	5.29	12.2	36	263	0.2	13	29	17.34	16*	0	78	6	0	0	68	23	34
7328	F	5.32	12.5	39	192	0.8	13	31	7.89	35	0	65	0	0	0	73	23	32
7383	F	5.04	13.5	42	120	0.4	13	28	8.22	47	0	48	4	1	0	83	27	32
Mean		5.22	13.0	40	210	0.5	13	36	11.61	36	0	60	4	0	0	76	25	33
30 mg/kg/day:																		
7367	M	4.98	12.4	38	143	0.2	12	28	10.84	41	0	57	2	0	0	76	25	33
7455	M	5.16	13.6	40	133	0.5	12	24	8.65	21	0	76	3	0	0	78	26	34
7382	F	4.84	12.8	38	157	0.6	13	26	5.83	26	0	73	1	0	0	79	26	34
7387	F	4.67	12.2	35	113	0.6	14	27	5.10	29	0	68	1	2	0	75	26	35
Mean		4.91	12.8	38	137	0.5	13	26	7.61	29	0	68	2	1	0	77	26	34
100 mg/kg/day:																		
7361	M	4.75	12.4	36	282	0.3	12	27	10.77	30	0	67	3	0	0	76	26	34
7456	M	5.36	13.4	42	196	0.2	11	28	5.84	38	0	60	0	1	1	78	25	32
7335	F	5.46	12.8	40	185	0.2	14	28	12.8	38	0	57	5	0	0	73	23	32
7381	F	4.82	11.5	36	115	0.5	14	26	10.36	54	0	44	1	0	1	75	24	32
Mean		5.10	12.5	39	195	0.3	13	27	9.58	40	0	57	2	0	1	76	25	33

*Repeat determination

[a] The differential leucocyte means have been adjusted to equal 100%.

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TABLE 6.

Individual Hematological Values - 1 Month.

Group, Monkey Number	Sex	Erythrocytes 10^6/cmm	Hemoglobin g/100 ml	Hematocrit %	Platelets 10^3/cmm	Reticulocytes %	Prothrombin Time sec	Activated P.T.T. sec	Leucocytes 10^3/cmm	Neutrophils Seg. %	Neutrophils Non-Seg. %	Lymphocytes[a] %	Eosinophils[a] %	Monocytes[a] %	Basophils[a] %	MCV μ^3	MCH µµg	MCHC g/100 ml
Control:																		
7362	M	4.80	11.9	38	224	0.2	12	30	6.91	28	0	69	3	0	0	79	25	31
7365	M	4.71	11.9	39	349	0.2	12	28	14.58	15	0	84	1	0	0	83	25	31
7336	F	4.20	11.2	37	246	0.2	13	28	7.46	11	0	89	0	0	0	88	27	30
7386	F	4.13	11.9	38	191	0.3	12	27	8.99	42	0	58	0	0	0	92	29	31
Mean		4.46	11.7	38	251	0.2	12	28	9.49	24	0	75	1	0	0	86	27	31
3 mg/kg/day:																		
7364	M	4.35	11.6	37	264	0.5	11	27	6.81	17	0	80	3	0	0	85	27	31
7366	M	3.96	10.7	35	188	0.4	12	28	5.83	16	0	78	6	0	0	88	27	31
7384	F	4.46	11.9	39	234	0.2	13	28	17.07	22	1	73	3	1	0	87	27	31
7385	F	4.25	11.2	35	247	0.9	12	29	9.41	18	0	73	9	0	0	82	26	32
Mean		4.26	11.4	37	233	0.5	12	28	9.78	19	0	76	5	0	0	86	27	31
10 mg/kg/day:																		
7363	M	4.42	12.1	38	168	1.0	13	27	8.08	42	0	57	1	0	0	86	28	32
7458	M	4.81	11.3	37	281	0.3	13	31	17.98	11	0	87	1	0	1	77	23	31
7328	F	4.70	12.0	39	181	0.5	13	33	7.01	35	0	63	2	0	0	83	26	31
7383	F	4.92	12.8	40	209	0.1	12	33	6.64	18	0	79	3	0	0	81	26	32
Mean		4.71	12.1	39	210	0.5	13	31	9.93	26	0	72	2	0	0	82	26	32
30 mg/kg/day:																		
7367	M	4.59	11.2	36	135	0.1	13	34	7.92	12	0	88	0	0	0	78	24	31
7455	M	4.44	11.8	37	237	0.2	14	33	11.11	27	0	73	0	0	0	83	27	32
7382	F	4.51	11.9	35	268	0.3	15	35	6.19	9	0	90	1	0	0	78	26	34
7387	F	4.56	12.0	37	237	0.2	16	38	8.54	11	0	87	0	0	0	81	26	32
Mean		4.53	11.7	36	219	0.2	15	35	8.44	15	0	85	0	0	0	80	26	32
100 mg/kg/day:																		
7361	M	Died, week 5																
7456	M	Died, week 2																
7335	F	Died, week 3																
7381	F	Died, week 4																

[a]The differential leucocyte means have been adjusted to equal 100%.

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TABLE 7.

Individual Hematological Values - 3 Months.

Group, Monkey Number	Sex	Erythrocytes 10^6/cmm	Hemoglobin g/100 ml	Hematocrit %	Platelets 10^3/cmm	Reticulocytes %	Prothrombin Time sec	Activated P.T.T. sec	Leucocytes 10^3/cmm	Neutrophils Seg. %	Neutrophils Non-Seg. %	Lymphocytes[c] %	Eosinophils[c] %	Monocytes[c] %	Basophils[c] %	MCV μ^3	MCH $\mu\mu g$	MCHC g/100 ml
Control:																		
7362	M	4.89	12.9	37	217	0.2	11	32	7.82	20	0	74	4	2	0	76	26	35
7365	M	5.29	13.1	37	218	0.3	10	25	12.84	10	0	85	4	1	0	70	25	35
7376	F	4.72	12.9	36	170	0.4	11	25	8.41	16	0	79	4	1	0	76	27	36
7386	F	4.69	12.8	36	234	0.3	11	20	8.51	18	1	80	0	1	0	77	27	36
Mean		4.90	12.9	37	210	0.3	11	26	9.40	16	0	80	3	1	0	75	26	36
3 mg/kg/day:																		
7364	M	4.86	12.9	37	299	0.1	11	24	7.33	24	0	71	4	1	0	76	27	35
7366	M	4.46	12.0	34	278	0.2	11	26	5.44	25	0	74	0	0	1	76	27	35
7384	F	4.92	13.0	39	313	0.2	11	28	18.21	16	0	76	5	3	0	79	26	33
7385	F	4.71	13.0	37	248	0.2	11	24	8.35	10	0	82	5	3	0	79	28	35
Mean		4.74	12.7	37	285	0.2	11	26	9.83	19	0	76	3	2	0	78	27	35
10 mg/kg/day:																		
7363	M	5.04	13.6	40	214	0.2	11	24	8.41	34	0	60	4	2	0	79	27	34
7458	M	5.70	12.6	40	218	0.3	11	23	20.18	4	0	94	2	0	0	70	22	32
7328	F	5.47	13.4	40	219	0.3	11	23	10.72	33	0	51	11	5	0	73	24	34
7383	F	5.65	13.5	39	212	0.1	11	27	8.52	30	0	64	5	1	0	69	24	35
Mean		5.47	13.3	40	216	0.2	11	24	11.96	25	0	67	6	2	0	73	24	34
30 mg/kg/day:																		
7367	M	Died, week 7																
7455	M	3.84[a,b]	9.7	30	261	0.2	30	65	10.14	36	0	54	3	7	0	78	25	32
7382	F	Died, week 13																
7387	F	Died, week 12																
Mean		3.84	9.7	30	261	0.2	30	65	10.14	36	0	54	3	7	0	78	25	32
100 mg/kg/day:																		
7361	M	Died, week 5																
7456	M	Died, week 2																
7335	F	Died, week 3																
7381	F	Died, week 4																

[a] 2+ Poikilocytosis
[b] 2 Nucleated erythrocytes/100 leucocytes
[c] The differential leucocyte means have been adjusted to equal 100%.

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TABLE 8. Means and Significance of Biochemical Values.

Biochemistry	Month of Study	Control	3 mg/kg/day	10 mg/kg/day	30 mg/kg/day
Glucose, mg/100 ml	1	89	117*	104	122
	3	81	96	88	66[a]
B.U.N., mg/100 ml	1	23.0	21.2	22.5	26.1
	3	27.6	20.2	22.0	22.6[a]
Alk. Phos., int'l units/l	1	597	847	601	365*
	3	851	783	743	360[a]
S.G.O.T., int'l units/l	1	29	35	34	59**
	3	45	41	35	88[a]
S.G.P.T. int'l units/l	1[b]	15	21	34*	44
	3[c]	31	31	34	46[a]
Cholesterol, mg/100 ml	1	165	154	158	174
	3	165	141	154	240[a]
Total Protein, g/100 ml	1	7.94	8.23	8.66	8.36
	3	8.21	8.24	8.43	5.52[a]
Albumin, g/100 ml	1	4.78	5.05	4.66	4.28
	3	4.82	5.12	5.17	2.00[a]
Sodium, meq/liter	1	153	152	155	152
	3	151	154	159**	150[a]
Potassium, meq/liter	1	5.1	5.1	5.2	5.7
	3	5.5	5.6	6.0	5.9[a]
Chloride, meq/liter	1	112	110	113	112
	3	113	112	114	113[a]
γ-G.T.P., Sigma units/ml	1	61	49	47	33
	3	44	38	51	49[a]
C.P.K., Sigma units/ml	1	9	14	16	19*
	3	7	6	9	10[a]
Inorganic Phosphate, mg/100 ml	1	7.9	7.2	7.0	6.7
	3	6.9	6.3	7.3	5.0[a]

*Significantly different from control group, $p<0.05$.
**Significantly different from control group, $p<0.01$.
[a]Value not used in statistical analysis due to only one animal surviving.
[b]I.U./l
[c]Sigma units/ml

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TABLE 9.

Individual Biochemical Values - Control 1.

Group, Monkey Number	Sex	Glucose mg/100 ml	B.U.N. mg/100 ml	Alk. Phos. Int'l units/l	S.G.O.T. Int'l units/l	S.G.P.T. Int'l units/l	Cholesterol mg/100 ml	Total Protein g/100 ml	Albumin g/100 ml	Sodium meq/l	Potassium meq/l	Chloride meq/l	Inorganic Phosphate mg/100 ml	γ-G.T.P. Sigma u/ml	Creatinine Phosphokinase Sigma u/ml
Control:															
7362	M	94	41.0	780	40	99	219	8.68	5.40	160	5.0	111	6.5	67	15
7365	M	82	16.7	659	61	88	123	9.50	4.30	155	5.3	110	6.7	44	18
7336	F	79	24.0	915	30	80	185	9.52	5.30	156	4.3	110	6.5	41	85
7386	F	85	21.0	960	39	86	190	8.52	5.12	162	5.0	111	6.5	37	16
Mean		85	25.7	829	43	88	179	9.06	5.03	158	4.9	111	6.6	47	34
3 mg/kg/day:															
7364	M	111	19.0	880	42	94	197	9.08	5.28	155	4.3	108	5.0	50	12
7366	M	71	28.7	580	60	89	172	9.12	5.80	157	4.9	108	7.1	30	26
7384	F	96	22.0	570	38	106	133	10.12	5.19	162	6.0	113	6.1	32	16
7385	F	107	22.0	1320	60	76	154	8.72	4.80	158	5.2	116	5.4	41	29
Mean		96	22.9	838	50	91	164	9.26	5.27	158	5.1	111	5.9	38	21
10 mg/kg/day:															
7363	M	89	27.2	1167	46	118	237	9.84	5.10	167	6.2	117	6.7	78	16
7458	M	180	24.2	806	63	136	107	10.08	3.99	150	4.9	107	7.7	55	14
7328	F	98	20.0	776	26	75	189	8.48	5.14	157	4.4	109	6.3	51	34
7383	F	98	27.3	581	31	91	168	8.32	5.25	159	5.1	112	6.0	59	64
Mean		116	24.7	833	42	105	175	9.18	4.87	158	5.2	111	6.7	61	32
30 mg/kg/day:															
7367	M	108	26.9	970	47	114	150	9.38	5.60	170	6.2	116	6.9	65	15
7455	M	110	24.0	687	37	86	205	9.50	5.31	163	5.3	111	6.6	59	9
7382	F	132	27.9	641	40	79	176	11.10	5.72	165	5.5	112	6.8	43	18
7387	F	117	23.8	978	45	138	194	9.44	5.60	155	3.9	113	5.4	39	16
Mean		117	25.7	819	42	104	181	9.86	5.56	163	5.2	113	6.4	52	15
100 mg/kg/day:															
7361	M	93	29.0	598	43	80	155	8.60	5.00	159	5.9	116	6.9	64	17
7456	M	100	23.0	799	40	104	202	9.00	5.69	157	4.5	109	5.7	44	22
7335	F	75	28.0	570	40	96	151	8.98	5.19	157	5.2	111	5.6	58	20
7381	F	119	22.1	1233	40	103	124	9.60	4.89	159	5.2	112	6.7	47	10
Mean		97	25.5	800	41	96	158	9.05	5.19	158	5.2	112	6.2	53	17

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TABLE 10.

Individual Biochemical Values - 1 Month.

Group, Monkey Number	Sex	Glucose mg/100 ml	B.U.N. mg/100 ml	Alk. Phos. Int'l units/l	S.G.O.T. Int'l units/l	S.G.P.T. Int'l units/l	Choles-terol mg/100 ml	Total Protein g/100 ml	Albumin g/100 ml	Sodium meq/l	Potas-sium meq/l	Chlo-ride meq/l	Inorganic Phosphate mg/100 ml	Y-G.T.P. Sigma u/ml	Creatinine Phosphokinase Sigma u/ml
Control:															
7362	M	87	33.9	611	27	18	191	7.30	4.82	153	5.4	117	6.6	81	8
7365	M	84	14.2	626	33	17	121	8.40	4.11	153	5.4	111	8.4	50	11
7336	F	87	23.9	672	25	15	142	7.90	4.89	148	4.2	109	8.4	68	7
7386	F	96	14.9	480	31	10	206	8.15	5.30	158	5.4	112	8.1	44	11
Mean		89	23.0	597	29	15	165	7.94	4.78	153	5.1	112	7.9	61	9
3 mg/kg/day:															
7364	M	112	18.0	970	30	36	173	8.15	5.20	150	4.3	106	6.9	77	4
7366	M	131	23.3	693	39	19	148	8.05	5.42	154	4.9	110	6.6	26	7
7384	F	105	24.2	539	30	15	141	8.70	4.85	152	5.8	111	7.5	47	39
7385	F	120	19.1	1185	40	13	153	8.00	4.72	152	5.2	114	7.8	47	7
Mean		117	21.2	847	35	21	154	8.23	5.05	152	5.1	110	7.2	49	14
10 mg/kg/day:															
7363	M	98	24.9	552	40	35	219	9.40	4.62	161	6.3	118	6.9	65	7
7458	M	97	22.5	732	40	43	134	9.05	4.32	151	4.9	109	8.4	44	20
7328	F	98	22.7	640	23	19	145	8.20	4.50	152	4.3	111	5.4	37	24
7383	F	124	20.0	480	31	37	132	8.00	5.19	154	5.2	113	7.2	43	14
Mean		104	22.5	601	34	34	158	8.66	4.66	155	5.2	113	7.0	47	16
30 mg/kg/day:															
7367	M	112	35.2	376	48	30	180	8.20	4.70	157	6.0	110	6.6	40	25
7455	M	86	21.0	322	61	80	177	8.55	3.22	148	5.2	112	6.9	40	16
7382	F	104	25.2	400	83	43	161	8.15	4.21	149	5.9	111	6.0	28	17
7387	F	185	22.8	360	45	23	179	8.55	5.00	153	5.6	114	7.2	24	18
Mean		122	26.1	365	59	44	174	8.36	4.28	152	5.7	112	6.7	33	19
100 mg/kg/day:															
7361	M	Died, week 5													
7456	M	Died, week 2													
7335	F	Died, week 3													
7381	F	Died, week 4													

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TABLE II. Individual Biochemical Values - 3 Months.

Group, Monkey Number	Sex	Glucose mg/100 ml	B.U.N. mg/100 ml	Alk. Phos. Int'l units/l	S.G.O.T. Int'l units/l	S.G.P.T. Sigma units/ml	Cholesterol mg/100 ml	Total Protein g/100 ml	Albumin g/100 ml	Sodium meq/l	Potassium meq/l	Chloride meq/l	Inorganic Phosphate mg/100 ml	γ-G.T.P. Sigma u/ml	Creatinine Phosphokinase Sigma u/ml
Control:															
7362	M	95	41.9	804	55	44	197	7.59	4.99	150	5.5	114	5.6	37	7
7365	M	77	17.4	744	47	30	135	9.18	4.40	151	6.1	113	8.0	53	8
7336	F	67	33.1	786	39	24	150	8.31	4.98	151	5.1	114	7.3	42	7
7386	F	86	18.1	1068	39	27	177	7.76	4.90	153	5.1	109	6.7	45	6
Mean		81	27.6	851	45	31	165	8.21	4.82	151	5.5	113	6.9	44	7
3 mg/kg/day:															
7364	M	106	17.1	1092	41	28	164	7.72	5.09	153	5.8	112	7.0	45	7
7366	M	111	18.1	594	39	33	126	8.09	5.52	153	5.5	109	5.3	51	6
7384	F	94	23.4	432	39	33	132	8.93	4.91	153	5.2	112	6.5	27	6
7385	F	74	22.0	1014	43	29	142	8.21	4.97	155	6.0	114	6.4	29	6
Mean		96	20.2	783	41	31	141	8.24	5.12	154	5.6	112	6.3	38	6
10 mg/kg/day:															
7363	M	87	24.9	936	42	42	194	8.44	5.61	164	7.0	119	8.0	43	7
7458	M	88	21.1	936	38	31	139	9.71	4.69	159	6.2	112	9.0	52	12
7328	F	75	21.8	624	30	25	155	7.93	5.27	156	4.8	110	5.6	60	7
7383	F	100	20.0	474	30	37	128	7.62	5.11	158	5.8	113	6.5	48	9
Mean		88	22.0	743	35	34	154	8.43	5.17	159	6.0	114	7.3	51	9
30 mg/kg/day:															
7367	M	Died, week 7													
7455	M	66	22.6	360	88	46	240	5.52	2.00	150	5.9	113	5.0	49	10
7382	F	Died, week 13													
7387	F	Died, week 12													
Mean		66	22.6	360	88	46	240	5.52	2.00	150	5.9	113	5.0	49	10
100 mg/kg/day:															
7361	M	Died, week 5													
7456	M	Died, week 2													
7335	F	Died, week 3													
7381	F	Died, week 4													

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TABLE 12. Means and Significance of Urinalysis Values.

Urinalysis	Month of Study	Control	3 mg/kg/day	10 mg/kg/day	30 mg/kg/day
Volume, ml	1	35	33	51	41
	3	71	94	51	40[a]
pH	1	8.5	8.5	8.1	8.1
	3	8.3	7.6	8.2	6.6[a]
Specific Gravity	1	1.028	1.026	1.026	1.026
	3	1.018	1.015	1.024	1.031[a]

[a]Value not used in statistical analysis due to only one animal surviving.

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TABLE 13.

Individual Urinalysis Values - Control 1.

Group, Monkey Number	Sex	Volume ml	Color and Appear.	pH	Spec. Grav.	Protein	Glucose	Occult Blood	Ketones	Leuco-cytes	Erythro-cytes	Epi. Cells	Urates	Triple Phos.	Cal. Oxal.	Uric Acid Crystals	Bacteria	Casts
Control:																		
7362	M	100	LS-cl	7.6	1.010	N	N	tr	N	-	occ	occ	F	occ	-	-	M	-
7365	M	28	LS-cl	7.2	1.037	N	N	N	N	-	1-3	occ	F	occ	-	-	M	-
7336	F	27	LS-C	7.0	1.036	N	N	N	1+	-	-	-	occ	occ	occ	-	F	-
7386	F	70	LS-cl	8.4	1.023	N	N	4+	N	-	-	occ	occ	occ	M	-	M	-
Mean		56		7.6	1.027													
3 mg/kg/day:																		
7364	M	25	LS-cl	7.8	1.032	N	N	tr	N	-	-	occ	F	F	F	-	M	-
7366	M	25	LS-cl	7.2	1.035	N	N	tr	N	-	-	occ	F	occ	occ	-	M	-
7384	F	215	LS-C	8.3	1.026	N	N	N	N	-	-	occ	occ	occ	-	-	M	-
7385	F	35	LS-cl	8.3	1.020	N	N	N	N	-	-	occ	F	occ	-	-	M	-
Mean		75		7.9	1.028													
10 mg/kg/day:																		
7363	M	20	LS-cl	7.7	1.020	N	N	tr	N	-	-	occ	F	F	-	-	M	-
7458	M	50	LS-cl	7.5	1.036	N	N	tr	N	-	-	occ	F	occ	F	-	M	-
7378	F	35	LS-cl	7.8	1.036	N	N	tr	N	-	-	1-3	F	occ	M	-	F	-
7383	F	35	LS-cl	8.2	1.020	N	N	3+	N	-	-	occ	occ	occ	-	-	F	-
Mean		35		7.8	1.028													
30 mg/kg/day:																		
7367	M	20	LS-cl	7.1	1.050	N	N	tr	N	-	1-3	1-3	occ	occ	occ	-	M	-
7455	M	35	LS-cl	6.8	1.030	N	N	tr	N	-	1-3	1-3	occ	F	-	-	M	-
7382	F	20	LS-cl	7.0	1.055	N	N	N	N	-	-	1-3	F	occ	-	-	F	-
7387	F	48	LS-cl	8.2	1.030	N	N	N	N	-	-	occ	F	occ	occ	-	M	-
Mean		31		7.3	1.041													
100 mg/kg/day:																		
7361	M	21	LS-cl	7.6	1.035	N	N	tr	N	-	occ	-	F	occ	-	-	M	-
7456	M	25	LS-cl	7.1	1.042	N	N	tr	3+	-	-	occ	F	occ	F	-	M	-
7335	F	25	LS-cl	7.2	1.041	N	N	tr	1+	-	1-3	-	occ	occ	F	-	F	-
7381	F	40	LS-cl	8.1	1.042	N	N	1+	1+	-	-	1-3	occ	occ	M	-	F	-
Mean		28		7.5	1.040													

Code:

tr - Trace
1+ - Trace to slight
2+ - Slight to moderate
3+ - Moderate
4+ - Marked

S - Straw
LS - Light Straw
DS - Dark Straw
LAm - Light Amber
DAm - Dark Amber
cl - Cloudy
C - Clear

N - Negative
F - Few
L - Loaded
M - Many
R - Rare
occ - Occasional

QNS - Quantity not sufficient
norm - Normal
- None seen

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Ninety Day Subacute Rhesus Monkey Toxicity Study.

TABLE 14. Individual Urinalysis Values - 1 Month.

Group, Monkey Number	Sex	Volume ml	Color and Appear.	pH	Spec. Grav.	Protein	Glucose	Occult Blood	Ketones	Leuco-cytes	Erythro-cytes	Epi. Cells	Urates	Triple Phos.	Cal. Oxal.	Uric Acid Crystals	Bacteria	Casts
Control:																		
7362	M	55	LS-C	8.5	1.021	N	N	N	N	-	occ	-	occ	occ	M	-	M	-
7365	M	35	LS-C	8.5	1.028	N	N	N	N	-	-	-	occ	F	occ	-	M	-
7336	F	20	LS-C	8.5	1.033	N	N	3+	N	-	-	1-3	F	F	F	-	M	-
7386	F	30	LS-C	8.5	1.030	N	N	tr	N	-	-	occ	M	F	M	-	M	-
Mean		35		8.5	1.028													
3 mg/kg/day:																		
7364	M	20	LS-C	8.8	1.019	N	N	N	N	-	-	occ	F	M	occ	-	M	-
7366	M	20	LS-C	8.5	1.036	N	N	N	N	-	-	occ	F	F	F	-	M	-
7384	F	40	DS-cl	8.0	1.021	1+	N	4+	2+	-	8-12	-	F	occ	F	-	M	-
7385	F	50	LS-cl	8.5	1.027	N	N	N	N	-	-	occ	F	occ	M	-	M	-
Mean		33		8.5	1.026													
10 mg/kg/day:																		
7363	M	65	LS-cl	7.5	1.023	N	N	N	N	-	occ	-	F	occ	M	-	M	-
7458	M	35	LS-C	8.0	1.028	N	N	N	N	-	-	-	occ	occ	M	-	M	-
7328	F	55	LS-cl	8.5	1.026	N	N	N	N	-	-	1-3	occ	occ	M	-	M	-
7383	F	50	LS-cl	8.5	1.028	N	N	tr	N	-	occ	occ	F	occ	M	-	M	-
Mean		51		8.1	1.026													
30 mg/kg/day:																		
7367	M	30	LS-C	7.5	1.024	N	N	N	N	-	-	occ	occ	occ	-	-	L	-
7455	M	30	LS-cl	8.0	1.026	N	N	N	N	-	occ	occ	M	F	-	-	M	-
7382	F	60	LS-cl	8.3	1.022	N	N	N	N	-	occ	-	F	F	-	-	M	-
7387	F	45	LS-cl	8.5	1.032	N	N	N	N	-	-	occ	F	F	occ	occ	M	-
Mean		41		8.1	1.026													
100 mg/kg/day:																		
7361	M	Died, week 5																
7456	M	Died, week 2																
7335	F	Died, week 3																
7381	F	Died, week 4																

Code:

tr - Trace
1+ - Trace to slight
2+ - Slight to moderate
3+ - Moderate
4+ - Marked

S - Straw
LS - Light Straw
DS - Dark Straw
LAm - Light Amber
DAm - Dark Amber
cl - Cloudy
C - Clear

N - Negative
F - Few
L - Loaded
M - Many
R - Rare
occ - Occasional

QNS - Quantity not sufficient
norm - Normal
- None seen

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TABLE 15. Ninety Day Subacute Rhesus Monkey Toxicity Study.

Individual Urinalysis Values - 3 Months.

Group, Monkey Number	Sex	Volume ml	Color and Appear.	pH	Spec. Grav.	Protein	Glucose	Occult Blood	Ketones	Leuco-cytes	Erythro-cytes	Epi. Cells	Urates	Triple Phos.	Cal. Oxal.	Uric Acid Crystals	Bacteria	Casts
Control:																		
7362	M	110	LS-C	8.2	1.012	N	N	N	N	-	-	occ	F	occ	-	-	M	-
7365	M	40	LS-cl	8.1	1.029	N	N	N	1+	-	occ	1-3	F	F	-	-	M	-
7336	F	85	LS-C	8.2	1.015	N	N	N	tr	-	-	occ	F	occ	F	-	M	-
7386	F	50	LS-C	8.8	1.016	N	N	3+	N	occ	-	occ	F	F	F	-	M	-
Mean		71		8.3	1.018													
3 mg/kg/day:																		
7364	M	50	LS-C	6.0	1.020	N	N	N	tr	-	-	-	F	occ	-	-	M	-
7366	M	150	LS-C	7.9	1.007	N	N	N	N	-	-	occ	F	occ	-	-	M	-
7384	F	125	LS-C	8.1	1.010	N	N	N	N	-	-	occ	F	F	F	-	M	-
7385	F	50	LS-C	8.5	1.021	N	N	tr	N	-	occ	1-3	M	F	M	-	M	-
Mean		94		7.6	1.015													
10 mg/kg/day:																		
7363	M	40	LS-C	8.0	1.027	N	N	N	N	-	-	occ	F	occ	occ	-	M	-
7458	M	35	LS-cl	8.7	1.022	N	N	N	N	-	-	-	F	occ	-	-	M	-
7328	F	50	LS-C	9.0	1.029	N	N	N	N	-	occ	occ	F	occ	-	-	M	-
7383	F	80	LS-cl	7.0	1.019	N	N	N	N	-	occ	occ	F	-	-	-	M	-
Mean		51		8.2	1.024													
30 mg/kg/day:																		
7367	M	Died, week 7																
7455	M	40	S-C	6.6	1.031	N	N	1+	N	1-3	occ	-	F	M	occ	-	M	-
7382	F	Died, week 13																
7387	F	Died, week 12																
Mean		40		6.6	1.031													
100 mg/kg/day:																		
7361	M	Died, week 5																
7456	M	Died, week 2																
7335	F	Died, week 3																
7381	F	Died, week 4																

Code: tr - Trace
1+ - Trace to slight
2+ - Slight to moderate
3+ - Moderate
4+ - Marked

S - Straw
LS - Light Straw
DS - Dark Straw
LAm - Light Amber
DAm - Dark Amber
cl - Cloudy
C - Clear

N - Negative
F - Few
L - Loaded
M - Many
R - Rare
occ - Occasional

QNS - Quantity not sufficient
norm - Normal
- None seen

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TABLE 16. Summary of Gross Necropsy Observations, Terminal Sacrifice.

Site / Lesion	0 mg/kg/day				3 mg/kg/day				10 mg/kg/day				30 mg/kg/day				100 mg/kg/day			
Group, Monkey Number	M 7362	M 7365	F 7136	F 7386	M 7364	M 7366	F 7384	F 7385	M 7363	M 7458	F 7328	F 7183	M* 7367	M 7455	F* 7382	F* 7187	M* 7361	M 7456	F* 7135	F* 7181
No Gross Lesions			x			=														
External																				
swelling, eye area														x						
alopecia																x				
dehydrated																x				
emaciated																x				
red vaginal discharge																x				
scab, facial area																x				
Lung																				
mite lesion	x	x		x		x		x		x	x	x	x							
adhesions		x						x		x		x		x	x					
dark red foci/reddish purple area							x				x			x		x				
yellow, white foci										x		x	x					x		x
nodules																	x	x		
Heart																				
hemorrhage, subendocardial																		x	x	
gelatinized fat, endocardial																			x	
atrophy																				x
Lymph Nodes																				
enlarged		x																		
reddish black in color																		x		
Thymus																				
atrophy														x						
Abdominal Cavity																				
depletion of fat																				x
Stomach																				
dark red foci										x				x		x				
erosion, mucosa, pyloric portion													x							
mucosal hyperemia														x						
yellowish gelatinous material, fundic portion																x				
hemorrhage, fundic mucosa																		x		
ulcers																			x	x
Small Intestine																				
greenish-gray mucoid material																				
dark red/brown mucoid material														x						
liquid, blood tinged fluid																	x		x	x
reddish brown in color																		x		
congestion, mucosa																		x		
hemorrhage, mucosa																			x	
Large Intestine																				
congestion, mucosa																				
hemorrhage, mucosa													x							
dark reddish black foci																	x			
semi solid, blood stained contents																	=	x		

*Died on Study

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FC-143: Ninety Day Subacute Rhesus Monkey Toxicity Study.

TABLE 16. Cont. Summary of Gross Necropsy Observations.

Site Lesion	Group, Monkey Number	0 mg/kg/day				3 mg/kg/day				10 mg/kg/day				30 mg/kg/day				100 mg/kg/day			
		M	M	F	F	M	M	F	F	M	M	F	F	M*	M	F*	F*	M*	M*	F*	F*
		7362	7365	7336	7386	7364	7366	7384	7385	7361	7458	7128	7383	7367	7455	7382	7387	7361	7456	7335	7381
Pancreas																					
accessory spleen									x												
Liver																					
cyst																					
brownish color												x									
accentuated lobulations															x						
granular surface															x				x		
yellowish mottling															x						
reddish yellow color																x			x		
Kidneys																					
brownish discoloration															x						
Skin																					
subcutaneous edema, abdomen																x					
subcutaneous hemorrhage, abdomen																			x		

*Died on Study

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Ninety Day Subacute Rhesus Monkey Toxicity Study.

TABLE 17. Absolute (Grams) and Relative (% Body Weight) Organ Weights, Terminal Sacrifice and Deaths.

Group, Monkey Number	Sex	Body Wt. kg	Spleen g	Spleen %	Liver g	Liver %	Adrenals g	Adrenals %x10	Kidneys g	Kidneys %	Testes/ Ovaries g	Testes/ Ovaries %x10^2
Terminal Sacrifice:												
Control:												
7362	M	1.25	2.35	0.07	70.73	2.18	0.65	0.20	11.82	0.36	0.85	0.03
7365	M	3.85	7.87	0.20	79.15	2.06	0.71	0.18	17.06	0.44	3.23	0.08
Mean		3.55	5.11	0.14	74.94	2.12	0.68	0.19	14.44	0.40	2.04	0.06
7336	F	3.40	5.03	0.15	84.79	2.49	-	-	13.80	0.41	0.28	0.82
7386	F	3.50	3.87	0.11	77.77	2.22	0.62	0.18	19.58	0.56	0.27	0.77
Mean		3.45	4.45	0.13	81.28	2.36	0.62[a]	0.18[a]	16.69	0.48	0.28	0.80
3 mg/kg/day:												
7364	M	4.10	4.67	0.11	91.40	2.23	0.77	0.19	19.76	0.48	3.66	0.09
7366	M	2.65	1.87	0.07	63.17	2.38	0.82	0.31	12.40	0.47	0.85	0.03
Mean		3.38	3.27	0.09	77.29	2.31	0.80	0.25	16.08	0.47	2.26	0.06
7384	F	3.70	6.82	0.18	102.64	2.77	0.78	0.21	17.60	0.48	0.18	0.49
7385	F	3.45	2.94	0.09	67.25	1.95	0.55	0.16	14.44	0.42	0.16	0.46
Mean		3.58	4.88	0.13	84.95	2.36	0.67	0.19	16.02	0.45	0.17	0.48
10 mg/kg/day:												
7363	M	3.80	2.39	0.06	87.25	2.30	0.74	0.19	16.84	0.44	1.75	0.05
7458	M	3.25	4.91	0.15	82.30	2.53	0.67	0.21	16.54	0.51	1.99	0.06
Mean		3.53	3.65	0.11	84.78	2.41	0.71	0.20	16.69	0.48	1.87	0.05
7328	F	3.55	4.06	0.11	83.00	2.34	0.66	0.19	15.32	0.43	0.29	0.82
7383	F	3.70	3.99	0.11	85.35	2.31	0.86	0.23	13.56	0.37	0.39	1.05
Mean		3.63	4.03	0.11	84.18	2.32	0.76	0.21	14.44	0.40	0.34	0.94
30 mg/kg/day[a]:												
7455	M	2.40	1.50	0.15	70.76	2.95	0.84	0.35	16.85	0.70	1.16	0.05
Deaths:												
30 mg/kg/day:												
7367	M	2.10	1.45	0.07	75.33	3.59	1.61	0.78	16.34	0.78	1.94	0.09
7382	M	2.25	3.01	0.13	112.87	5.02	1.74	0.77	19.03	0.85	0.21	0.93
7387	F	2.25	1.97	0.09	85.17	3.79	1.20	0.53	15.96	0.71	0.32	1.42
100 mg/kg/day:												
7361	M	2.40	1.65	0.07	79.02	3.29	1.59	0.66	21.88	0.91	1.37	0.06
7456	M	2.70	1.76	0.07	85.08	3.15	1.45	0.54	14.77	0.55	0.71	0.03
7335	F	2.05	2.49	0.12	74.28	3.62	1.03	0.50	15.40	0.75	0.10	0.51
7381	F	2.60	3.05	0.12	82.58	3.18	1.16	0.45	18.28	0.70	0.13	0.50

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Group mean relative organ weights shown in this table were calculated by averaging the individually calculated relative organ weights.

*Significantly different from Control group mean, p<0.05.

**Significantly different from Control group mean, p<0.01.

[a]Not included in analysis.

- = Not available

FC-141:

Ninety Day Subacute Rhesus Monkey Toxicity Study.

TABLE 17. Cont. Absolute (Grams) and Relative (% Body Weight) Organ Weights, Terminal Sacrifice and Deaths.

Group, Monkey Number	Sex	Body Wt. kg	Heart g	Heart %	Thyroid/ Parathyroid g	Thyroid/ Parathyroid %x10	Brain g	Brain %	Pituitary g	Pituitary %x10[2]
Terminal Sacrifice:										
Control:										
7362	M	3.25	11.69	0.36	1.050	0.32	87.04	2.68	0.053	0.16
7365	M	3.85	18.17	0.47	0.296	0.08	90.39	2.35	0.063	0.16
Mean		3.55	14.93	0.42	0.673	0.20	88.72	2.51	0.058	0.16
7376	F	3.40	15.30	0.45	-	-	82.64	2.43	0.050	0.15
7386	F	3.50	14.75	0.42	0.839	0.24	81.55	2.33	0.073	0.21
Mean		3.45	15.03	0.44	0.839[a]	0.24[a]	82.10	2.38	0.062	0.18
3 mg/kg/day:										
7364	M	4.10	18.90	0.46	0.893	0.22	96.01	2.34	0.080	0.20
7366	M	2.65	12.70	0.48	0.378	0.14	83.50	3.15	0.051	0.19
Mean		3.38	15.80	0.47	0.636	0.18	89.76	2.75	0.066	0.19*
7384	F	3.70	16.87	0.46	0.694	0.19	78.66	2.13	0.086	0.23
7385	F	3.45	15.19	0.44	0.543	0.16	80.21	2.32	0.053	0.15
Mean		3.58	16.03	0.45	0.619	0.17	79.44	2.23	0.070	0.19
10 mg/kg/day:										
7363	M	3.80	15.10	0.40	1.211	0.32	77.73	2.05	0.063	0.17
7458	M	3.25	14.14	0.44	0.488	0.15	83.38	2.57	0.047	0.14
Mean		3.53	14.62	0.42	0.850	0.23	80.56	2.31	0.055	0.16
7328	F	3.55	11.85	0.33	0.461	0.13	77.19	2.17	-	-
7381	F	3.70	11.69	0.32	0.537	0.15	75.88	2.05	0.071	0.19
Mean		3.63	11.77*	0.32**	0.499	0.14	76.54**	2.11	0.071[a]	0.19[a]
30 mg/kg/day[a]:										
7455	M	2.40	10.50	0.44	0.292	0.12	75.01	3.13	0.049	0.20
Deaths:										
30 mg/kg/day:										
7367	M	2.10	10.39	0.49	0.532	0.25	82.27	3.92	0.068	0.32
7382	F	2.25	11.93	0.53	0.543	0.24	83.22	3.70	0.070	0.31
7387	F	2.25	10.21	0.45	0.845	0.38	91.45	4.06	0.057	0.25
100 mg/kg/day:										
7361	M	2.40	14.54	0.61	0.791	0.33	92.43	3.85	0.072	0.30
7456	M	2.70	15.55	0.58	0.718	0.27	95.42	3.53	0.046	0.17
7335	F	2.05	11.44	0.56	0.479	0.23	74.28	3.62	0.056	0.27
7381	F	2.60	12.95	0.50	0.417	0.16	86.20	3.32	0.082	0.32

Group mean relative organ weights shown in this table were calculated by averaging the individually calculated relative organ weights.

*Significantly different from Control group mean, $p \leq 0.05$.

**Significantly different from Control group mean, $p \leq 0.01$.

[a] Not included in analysis.

- = Not available

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FC-143: Ninety Day Subacute Rhesus Monkey Toxicity Study.

TABLE 18. Microscopic Observations.

Tissue Lesion	Control				3 mg/kg/day				10 mg/kg/day				30 mg/kg/day				100 mg/kg/day			
Sex	M	M	F	F	M	M	F	F	M	M	F	F	M	M	F	F	M	M	F	F
Group, Monkey Number	7362	7365	7336	7386	7364	7366	7384	7385	7363	7458	7328	7383	7455	7367*	7382*	7387*	7456*	7361*	7335*	7381*
Brain	1	1	1	1	1	1	1	1	1		1	1	1	1	1	1	1	1	1	1
focal perivascular lymphoid infiltrates										3										
Spinal cord	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Peripheral nerve	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Eye	1		1	1	1	1						1		1	1	1	1	1		1
Sarcocystis sp. in ocular muscle		x					x												x	
focal lymphoid infiltrates in sclera											3									
focal lymphoid infiltrates in lacrimal gland								3					3							
focal lymphoid infiltrate in palpebral conjunctiva									3	3										
cystic tarsal gland										3										
Pituitary	1	1	1	1	1	1	1	1	1		1	1	1		1			1		
diffuse congestion										x				3		3	3		3	3
small parenchymal cyst																				
Thyroid	1	1	1		1		1	1	1	1	1	1	1	1				1	1	
foci of interstitial lymphoid infiltrates				3		2										2				
focal interstitial fibrosis				3												2				
diffuse congestion															3		3			3
Parathyroid	1	1	1	1	1	1	-	-	-	-	-	-	-	1	-	-		-	-	1
diffuse congestion																	3			
Tongue	1								1			1			1	1	1	1		
foci of inflammatory cell infiltrates in lamina propria and mucosal epithelium		3	3	4	2	3	2	3		3	3		2	2					2	2
foci of inflammatory cell infiltrates in muscle		2					3			3	2		2						2	
Sarcocystis sp.							x				x									

Code:
x - condition present
a - autolyzed
1 - not remarkable
2 - very slight
3 - slight
4 - moderate
5 - marked
6 - extreme
- = not available
*Died or sacrificed in extremis

FC-143: Ninety Day Subacute Rhesus Monkey Toxicity Study.

TABLE 18. Cont. Microscopic Observations.

Tissue Lesion	Group, Sex Monkey Number	Control M 7362	Control M 7365	Control F 7336	Control F 7386	3 mg/kg/day M 7364	3 mg/kg/day M 7366	3 mg/kg/day F 7384	3 mg/kg/day F 7385	10 mg/kg/day M 7363	10 mg/kg/day M 7458	10 mg/kg/day F 7328	10 mg/kg/day F 7383	30 mg/kg/day M 7455	30 mg/kg/day M 7367*	30 mg/kg/day F 7382*	30 mg/kg/day F 7387*	100 mg/kg/day M 7456*	100 mg/kg/day M 7361*	100 mg/kg/day F 7335*	100 mg/kg/day F 7381*
Tonsil																					
foci of inflammatory cell infiltrates in mucosal epithelium and tonsillar crypt		3	4	2	3	1	4	3	3	3	3	4	4	-	2	1	3	-	-	-	4
Sarcocystis sp. in muscle			x																		
Gongylonema sp. in mucosal epithelium					x												4				4
atrophy of lymphoid follicles																					
Adrenal																					
foci of dystrophic mineralization		3	3	2	2	3	1	2			3	2	2					2			
diffuse congestion															3	4	3	3		4	3
diffuse lipid depletion															5	5	5	5	5	5	5
foci of lymphoid infiltrates in sinusoids				3		2		2	3	3	3		2								
acidophilic degeneration of individual to small groups of cells														2			3				
Trachea																					
foci of inflammatory cell infiltrates in lamina propria		3	1	3	3	3	2	2	3	3	3	3	2	2	1	3	1	1	1	3	3
Salivary gland																					
focal interstitial lymphoid infiltrates		2	3	1	2	1	3	4	3	1	2	2	3	3	3	2	3	3	1	1	
diffuse congestion																3					
decreased cell size, loss of cytoplasmic granules															4			4			3
Lung																					
acarian pigment (peribronchial, peribronchiolar, perivascular)		3	2	2	2	3	2	2	2	2	2	3	2	3	2	2	4	2		2	2
focal perivascular lymphoid infiltrates						3					3	3									
focal peribronchial/peribronchiolar lymphoid aggregates		4	4	3	4	3	3	4	3	3	4	4	3	3		2	2			3	3
lung mite in bronchiolar lumen		x			x																
interstitial pneumonia		3	4		4	3		3	4	3			3	4		4			3		
diffuse congestion															3	3	3	4			
foreign body pneumonia			5						5												
focal hemorrhage			3																		
acute focal bronchopneumonia		4				3					4							3			
numerous aggregates of pigment laden alveolar macrophages																5					

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*Died or sacrificed in extremis

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FC-143: Ninety Day Subacute Rhesus Monkey Toxicity Study.

TABLE 18. Cont. Microscopic Observations.

Tissue Lesion	Group, Sex Monkey Number	Control M 7362	Control M 7365	Control F 7336	Control F 7386	3 mg/kg/day M 7364	3 mg/kg/day M 7366	3 mg/kg/day F 7384	3 mg/kg/day F 7385	10 mg/kg/day M 7363	10 mg/kg/day M 7458	10 mg/kg/day F 7328	10 mg/kg/day F 7383	30 mg/kg/day M 7455	30 mg/kg/day M 7367*	30 mg/kg/day F 7382*	30 mg/kg/day F 7387*	100 mg/kg/day M 7456*	100 mg/kg/day M 7361*	100 mg/kg/day F 7335*	100 mg/kg/day F 7381*
Heart			1			1				1	1		1	1		1			1		
focal interstitial lymphoid infiltrates		3		3	3		2	3	3						3			2		2	
focus of lymphoid infiltrate in endocardium												3						3			
focal subendocardial hemorrhage																	4			4	4
atrophy of epicardial fat																					
Aorta		1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Spleen		1	1	1	1	1	1	1	1	1	1										
atrophy of lymphoid follicles												3	3	4 3	4 3	4 4	4 3	4 4	4 4	4 3	4 4
diffuse congestion																				3	
focal amyloidosis in lymphoid follicles																	3				
increased amount of hemosiderin pigment																					
Lymph node		1		1	1	1	1	1	1	1	1	1	1	1	1						
atrophy of lymphoid follicles			3														4	4	4	4	4
increased amount of hemosiderin pigment																		3			
neutrophil infiltrate in sinuses																3			3	5	
diffuse congestion			3													3					3
lymphoid hyperplasia																					
Esophagus		1			1		1									1		1		1	1
foci of inflammatory cell infiltrates in lamina propria			3	2		2		3	2		3	2	2	3	2		2		2		
foci of interstitial lymphoid infiltrates in muscularis			2					2			2	2	2								
Gongylonema sp. in mucosal epithelium										x											
Stomach		3	4	3	3	3	3	4	4	4	3	4	3	3			3	3	2	4	3
foci of inflammatory cell infiltrate in lamina propria												2			3	3				3	
diffuse congestion																					
foci of inflammatory cell infiltrates in submucosa					4				4		4	3									
foci of inflammatory cell infiltrates in muscularis							3			3											
foci of inflammatory cell infiltrates in serosa										3											
parasitic granuloma in omentum										x											
focal mucosal hemorrhage											2		2							2	
focal coagulation necrosis in mucosa																				3	

Code: x - condition present; a - autolyzed; 1 - not remarkable; 2 - very slight; 3 - slight; 4 - moderate; 5 - marked; 6 - extreme; - = not available; *Died or sacrificed in extremis

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FC-143: Ninety Day Subacute Rhesus Monkey Toxicity Study.

TABLE 18. Cont. Microscopic Observations.

Tissue / Lesion	Group, Sex, Monkey Number	Control M 7362	Control M 7365	Control F 7336	Control F 7386	3 mg/kg/day M 7364	3 mg/kg/day M 7366	3 mg/kg/day F 7384	3 mg/kg/day F 7385	10 mg/kg/day M 7363	10 mg/kg/day M 7458	10 mg/kg/day F 7328	10 mg/kg/day F 7383	30 mg/kg/day M 7455	30 mg/kg/day M 7367*	30 mg/kg/day F 7382*	30 mg/kg/day F 7387*	100 mg/kg/day M 7456*	100 mg/kg/day M 7361*	100 mg/kg/day F 7335*	100 mg/kg/day F 7381*
Small intestine		1	1	1	1	1	1	1	1	1	1	1	1	1							
diffuse villous atrophy																					
focal hemorrhage																		5	5		
diffuse congestion															3				3	3	
focal aggregate of brown pigment-laden foamy macrophages in mesentery															3	3	3			3	3
inflammatory cell infiltrates in serosa																					x
atrophy of lymph nodule															4			4		4	
Cecum		1	1	-	1	1	1	1	1		1	1	1					1			1
transmural inflammatory cell infiltrates																					
diffuse congestion																			4		
focal mucosal hemorrhage															3	3	3		3	3	
inflammatory cell infiltrates in serosa															2				2	4	
parasitic granuloma in muscularis										2											
atrophy of lymph nodule														x							
																	4			4	
Colon		1	1	1	1	1	1	1	1	1	1	1	1	1							1
diffuse congestion															3	3	3			3	3
parasitic granuloma in submucosa																		x			
transmural inflammatory cell infiltrates																		4			
focal mucosal hemorrhage															3						
atrophy of lymph nodule																	4			4	
Rectum		1	1	1	1	1	1	1	1	1	1	1	1	1				-	1		1
diffuse congestion															3	3	3			3	
inflammatory cell infiltrates in muscularis																				3	
atrophy of lymphoid nodule																	4			4	
Pancreas		1	1				1			1		1	1					a	1	1	a
focal periductal lymphoid infiltrates				3	2	3		3			3			2							
focal interstitial lymphoid infiltrates								3	2												
diffuse congestion														3	3	3					
Thymus		1	1	1	1	1	1	1	1	1	1	1	1	-	-	-	-	-	-	-	-

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FC-143: Ninety Day Subacute Rhesus Monkey Toxicity Study.

TABLE 18. Cont. Microscopic Observations.

Tissue Lesion	Group, Sex Monkey Number	Control M 7362	Control M 7365	Control F 7336	Control F 7386	3 mg/kg/day M 7364	3 mg/kg/day M 7366	3 mg/kg/day F 7384	3 mg/kg/day F 7385	10 mg/kg/day M 7363	10 mg/kg/day M 7458	10 mg/kg/day F 7328	10 mg/kg/day F 7383	30 mg/kg/day M 7455	30 mg/kg/day M 7367*	30 mg/kg/day F 7382*	30 mg/kg/day F 7387*	100 mg/kg/day M 7456*	100 mg/kg/day M 7361*	100 mg/kg/day F 7335*	100 mg/kg/day F 7381*
Liver																					
portal inflammatory cell infiltrates		3	3	3	3			3	2	3	3	2	2		2				1		
parenchymal inflammatory cell infiltrates		2	2	2	3	3	3	3	3	3	3	2			4	3	3	3		3	2
diffuse congestion																		3		3	3
acidophilic degeneration of individual to small groups of hepatocytes									3								3				
diffuse hepatocellular hypertrophy with cytoplasmic vacuolation														3		3					
neutrophil infiltrates in sinusoids														3							
Gallbladder																					
foci of inflammatory cell infiltrates in lamina propria		3	3	4	3	3	2	2	3	2	3	3	3	1	a	a	a	a	a	1	a
Kidney																					
focal interstitial lymphoid infiltrates		2	2		2	3	3	4	2	2	3	2	3	2		2	2		2	2	
multinucleated lining epithelium in papillary ducts			x	x				x					x								
cyst in medulla			x																		
chronic interstitial nephritis				3																	
diffuse congestion															4	3	3	3	3	3	3
microlith in renal tubules																		x			
small foci of dystrophic mineralization				2										2			2		2	2	
Urinary bladder																					
foci of inflammatory cell infiltrates in lamina propria		3	2	3	2	2	3	2	3	3	3	1	1	1	1		1		1		
diffuse congestion																3		3		3	3
Testes																					
prepuberal development		x	x			x	x			x	x			x	x			x	x		
chronic focal vasculitis			4																		
focal perivascular lymphoid infiltrate											2										
Ovaries																					
small foci of dystrophic mineralization				1			1	1			1	1				1			1	1	
diffuse congestion			2												2						
															3						

Code: x - condition present; a - autolyzed; 1 - not remarkable; 2 - very slight; 3 - slight; 4 - moderate; 5 - marked; 6 - extreme; - = not available; *Died or sacrificed in extremis

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FC-143: Ninety Day Subacute Rhesus Monkey Toxicity Study.

TABLE 18. Cont. Microscopic Observations.

Tissue Lesion	Group, Sex, Monkey Number	Control M 7362	Control M 7365	Control F 7336	Control F 7386	3 mg/kg/day M 7364	3 mg/kg/day M 7366	3 mg/kg/day F 7384	3 mg/kg/day F 7385	10 mg/kg/day M 7363	10 mg/kg/day M 7458	10 mg/kg/day F 7328	10 mg/kg/day F 7383	30 mg/kg/day M 7455	30 mg/kg/day M 7367*	30 mg/kg/day F 7382*	30 mg/kg/day F 7387*	100 mg/kg/day M 7456*	100 mg/kg/day M 7361*	100 mg/kg/day F 7335*	100 mg/kg/day F 7381*
Prostate																					
focal interstitial lymphoid infiltrates		3	3			2	3			2	3			2	1			1	-		
focal lymphoid infiltrate in corpus cavernosum			3				2			2				3							
Uterus																					
diffuse congestion												1	1				1				
blood in uterine glands				2	2			2								3				3	3
small foci of hemorrhage in endometrium				2	2			3								2				2	
brown pigment-laden macrophages in endometrium									3												
inflammatory cell infiltrates in endometrium				3	2			4	2												
proteinaceous fluid and inflammatory cells in uterine lumen																				3	
Vagina																					
foci of lymphoid infiltrates in lamina propria and mucosal epithelium				3	4			3	3			4	4			2	3			2	5
foci of lymphoid infiltrates in muscularis					2				2				3								3
Sarcocystis sp.								x													
focal lymphoid infiltrate in tunica adventitia								3													
diffuse congestion																3					
focal neutrophil infiltrate in mucosa																					
Skeletal muscle		1		1	1	1	1			1		1						1			
Sarcocystis sp.			x					x	x						x					x	
focal interstitial inflammatory cell infiltrates			3				4		2		3		2								
interstitial fibrosis																4	4				3
focal/multifocal atrophy of muscle														4	3			4			4
increased sarcolemmal nuclei																					
Skin																					
brown/black pigment in dermis		x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x
dermal inflammatory cell infiltrates			2			3	3														
diffuse acanthosis		3		3																	
diffuse congestion																					
hyperkeratosis					3	3		3		3	3			3	3	3		3		3	3
few large areas of hemorrhage in subcutis							3										5				

Code: x - condition present; a - autolyzed; 1 - not remarkable; 2 - very slight; 3 - slight; 4 - moderate; 5 - marked; 6 - extreme; - = not available; *Died or sacrificed in extremis

137-090

FC-143: Ninety Day Subacute Rhesus Monkey Toxicity Study.

TABLE 18. Cont. Microscopic Observations.

Tissue Lesion	Control				3 mg/kg/day				10 mg/kg/day				30 mg/kg/day				100 mg/kg/day			
	M	M	F	F	M	M	F	F	M	M	F	F	M	M	F	F	M	M	F	F
	7362	7365	7336	7386	7364	7366	7384	7385	7363	7458	7328	7383	7455	7367*	7382*	7387*	7456*	7361*	7335*	7381*
Mammary gland																				
brown pigment in dermis	x	x		x	x			1	x	x	x		x	x		x	x	x	x	x
hyperkeratosis	3		3	3	3	3	3			3		3	3	3	3					
dermal inflammatory cell infiltrates			3	3	2		3		3		3	3	2							
inflammatory exudate in acinar lumen/ducts		2		2												2				
inflammatory cell infiltrates in intralobular connective tissue		3							2								3			
diffuse congestion													x							
intraepidermal microabscess																				
Femur	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	1	1	1	1	1
Bone marrow (Rib junction)	1	1	1	1	1	1	1	1	1	1	1	1								
hypocellular marrow													3	4	4	3	4	4	4	4
congestion														3	3	4	3	3	4	3
Miscellaneous																				
acute focal cheilitis, lip																4				

Code: x - condition present
a - autolyzed
1 - not remarkable
2 - very slight
3 - slight
4 - moderate
5 - marked
6 - extreme
- = not available
*Died or sacrificed in extremis

137-090

Exhibit 4

FC-95, FC-143 and FM-3422 - 90 Day Subacute Toxicity Studies

Conducted at IRDC - Review of Final Reports and Summary

OVERALL SUMMARY AND RECOMMENDATIONS

FC-95 was the most toxic of the three compounds studied and certainly more toxic than anticipated. It produced mortalities in rats at a dietary dose of 100 ppm (≈10 mg/kg/day) and in monkeys at an oral dose of 4.5 mg/kg/day. The primary target organs in rats were the liver, hematopoietic tissues and upper gastrointestinal tract and in monkeys, the gastrointestinal tract although no pathological lesions were reported. FC-143 appeared to be the least toxic of the three compounds studied and produced no mortalities in rats at dietary doses as high as 1000 ppm (≈ 100 mg/kg/day). However, definite evidence of liver toxicity was seen at the high dose. In monkeys, FC-143 caused deaths at oral doses of 100 (4/4) and 30 (3/4) mg/kg/day and evidence of effects on hematopoietic tissue at these lethal doses. Like FC-95 and FM-3422, FC-143 also produced clinical evidence of gastrointestinal toxicity but no associated pathological lesions. FM-3422 caused deaths in rats at dietary doses of 1000, 3000 and 10,000 ppm (≈ 100, 300 and 1000 mg/kg/day respectively) and in monkeys (1/4) at an oral dose of 30 mg/kg/day. The primary target organ in rats appeared to be the liver although there was some gross evidence of kidney and upper gastrointestinal tract involvement as well. In monkeys, the gastrointestinal tract was affected clinically, but there were no pathological lesions reported at necropsy.

The goals of conducting these 90 day subacute toxicity studies of 1) defining doses for chronic experiments and 2) obtaining general toxicological information on the three compounds appear to have been met. However, several questions surfaced that deserve further clarification. The apparent effect of FC-95 on the liver and hematopoietic system of rats should be studied for reversibility. The question of clinical gastroinestinal signs in monkeys with all three compounds without any gross or microscopic pathology is certainly perplexing, but may not be worth further pursuit since the oral route is not a likely one for man. If another study with FC-143 is conducted to help define the gastrointestinal and hematopoietic effects, the dog should be considered. Since the most likely route of exposure in plant workers is by inhalation, an inhalation study, probably with FM-3422, could be useful in evaluating any effects via pulmonary exposure. Marv Case and Bill McCormick are preparing protocols for follow-up to the toxicity questions mentioned.

Because of the apparent persistence of these fluorochemicals in the body, the most important question remains possible long term effects. Although lifetime rodent studies have limitations in predicting chronic effects (carcinogenesis) for man, they are still considered the most reliable indicators available. Unless there are adequate data through human epidemiological evaluations that can reasonably assure relative safety of these compounds following long term exposure, lifetime rodent studies should be undertaken as soon as possible. It may be possible to limit the number of compounds evaluated in lifetime rodent studies to one or two if metabolic data can be used to establish a common linkage between compounds.

INDIVIDUAL SUMMARIES

FC-95

Study No. 137-085 - 90 Day Subacute Rat Toxicity Study

Dietary levels of FC-95 were administered to five male and five female rats/level at 30, 100, 300, 1000 and 3000 ppm which approximates 3, 10, 30, 100 and 300 mg/kg/day respectively. All rats at the three highest doses and 5/10 at 100 ppm died during the study. Predominant signs observed included emaciation, convulsions, altered posture, ocular, oral and anal discharges, hyperreactivity and reduced motor activity. Mortalities occurred in a sequence related to dose, with earlier deaths seen at the highest level. There was compound and dose related evidence of reduced body weight gain and food consumption with actual weight loss at higher lethal doses. At 30 ppm only slight body weight effects were present. The most notable clinical pathology effects were observed at 100 ppm (values not obtained at higher levels) and consisted of enzyme level increases suggestive of possible liver toxicity and decreased erythrocytic values (principally hemoglobin and hematocrit with slight lowering of red cell counts) indicating an anemia. Pathologically, the most consistent and apparent compound related effect involved liver, hematopoietic tissues (thymus, bone marrow, spleen, mesenteric lymph nodes), gastrointestinal tract, muscle and skin.

In summary, FC-95 was relatively toxic to rats causing mortalities at dietary doses as low as 100 ppm ($\approx$ 10 mg/kg/day). Primary target organs appeared to be liver, hematopoietic tissues, stomach and small intestine with some indication of a compound related effect in muscle and skin.

Study No. 137-087 - 90 Day Subacute Rhesus Monkey Toxicity Study

FC-95 was administered by gastric intubation as an aqueous suspension to two male and two female rhesus monkeys/level at doses of 10, 30, 100 and 300 mg/kg/day for up to 20 days. Because of unexpected early mortalities in all monkeys at all levels (days 2-4 at 300, 3-5 at 100, 7-10 at 30 and 11-20 at 10 mg/kg/day), the study was inconclusive. Prominent signs observed consisted of anorexia, decreased activity, emesis with some diarrhea, body stiffening, general body trembling and twitching, weakness, convulsions and prostration. No clinical pathology work was done because of the short study duration. The only pathological lesions reported consisted of gross yellowish-brown liver coloration at 100 and 300 mg/kg/day but no histopathologic basis for this finding was observed.

In summary, FC-95 proved to be considerably more toxic to monkeys than anticipated resulting in early deaths preceded by gastroinestinal and central nervous system signs. Although far from definitive, this study suggested the gastrointestinal tract and possibly liver as target organs.

Study No. 137-092 - 90 Day Subacute Rhesus Monkey Toxicity Study (Second Study)

Since all monkeys died in the first FC-95 study (137-087), a second experiment was conducted using oral gavage doses of 0.5, 1.5 and 4.5 mg/kg/day administered to two male and two female monkeys/dose. The controls were the same monkeys used in the first FC-95 experiment. All 4.5 mg/kg monkeys exhibited signs of gastrointestinal tract toxicity (anorexia, emesis, black stools, dehydration) starting on day 1 or 2 of the study, and all died or were sacrificed in extremis between weeks 5-7. Prior to death, these monkeys showed marked or severe rigidity, convulsions, general body tremors, prostration and loss of body weight. The monkeys at lower doses all survived, but evidence of gastrointestinal toxicity was observed both at 1.5 and 0.5 mg/kg/day. The only consistent clinical pathology observation reported was decreased alkaline phosphatase values at all three doses. No gross pathological lesions considered compound related were observed and the only microscopic pathology of apparent compound relationship consisted of lipid depletion in the adrenals, atrophy of pancreatic exocrine cells and atrophy of the serous alveolar cells of the submandibular salivary glands in high dose monkeys. These latter effects may be due to general debilitation of the animals.

In summary, FC-95 was relatively toxic to rhesus monkeys producing deaths at doses as low as 4.5 mg/kg/day in 5-7 weeks. The apparent target organ was the upper gastrointestinal tract although no pathological lesions were reported even at the high dose.

FC-143

Study No. 137-089 - 90 Day Subacute Rat Toxicity Study

Dietary levels of FC-143 administered to five male and five female rats/level were 10, 30, 100, 300 and 1000 ppm which approximates 1, 3, 10, 30 and 100 mg/kg/day respectively. Clinically, the only effect observed was slightly decreased body weight gains at 300 and 1000 ppm. Clinical pathology abnormalities reported in high dose male rats only included slightly lowered erythrocyte counts, and elevated BUN and alkaline phosphatase values. There were several other variations from control groups in the clinical pathology parameters including fairly consistent lowering of calcium levels at all doses, but these were not considered abnormal based on the contract laboratory's comparison to background control data. Pathological abnormalities were confined to the liver and included gross enlargement and discoloration at 1000 ppm, increased organ weights at 1000 and 300 ppm and several microscopic changes at 1000 ppm.

In summary, FC-143 was well tolerated in rats at doses up to and including 300 ppm (≈ 30 mg/kg/day). There was obvious liver toxicity at 1000 ppm (≈ 100 mg/kg/day), but no mortalities occurred.

Study No. 137-090 - 90 Day Subacute Rhesus Monkey Toxicity Study

FC-143, suspended in 0.5% methocel, was administered by gastric intubation to two male and two female rhesus monkeys/dose at 3, 10, 30 and 100 mg/kg/day. All high dose monkeys died during weeks 2-5 and 3/4 30 mg/kg monkeys died during the last half of the study. All monkeys that died showed clinical evidence of gastrointestinal toxicity (anorexia, emesis, dark stools), but there were no associated pathological lesions found at necropsy. No mortalities occurred and only occasional signs of gastrointestinal effects were reported at the two lower doses except for one 10 mg/kg monkey that had signs of gastrointestinal toxicity for several days late in the study. There were a few abnormalities reported in clinical pathology parameters, but no consistent pattern was observed. Gross and microscopic pathological lesions were restricted to the two highest dose levels and consisted of lipid depletion in adrenals, hypocellularity of bone marrow and atrophy of lymphoid follicles of the spleen and lymph nodes.

In summary, FC-143 was less toxic than FC-95 in rhesus monkeys but, at lethal doses (100 and 30 mg/kg/day), evidence of effects on hematopoietic tissue was seen. Like FC-95, the gastrointestinal tract also appeared to be a target organ although this was not confirmed on histopathological examination.

FM-3422

Study No. 137-086 - 90 Day Subacute Rat Toxicity Study

FM-3422 was administered in the diet to five male and five female rats/level at 30, 100, 300, 1000, 3000 and 10,000 ppm which corresponds to approximately 3, 10, 30, 100, 300 and 1000 mg/kg/day respectively. All rats at the 1000, 3000 and 10,000 ppm levels died between days 9 and 29. Prominent signs observed in these rats included emaciation, altered posture, convulsions, reduced motor activity and/or increased sensitivity. At 30 ppm, a slight decrease in body weight gain in females was the only clinical effect reported. There were also some slight abnormalities in serum enzyme levels, but no pronounced trends. Likewise, minimal effects were seen at 100 ppm. At 300 ppm there appeared to be increased compound related clinical signs, decreased body weight gain and food consumption, depressed hematological parameters and several alterations in clinical chemistry values. Pathologically, the liver was grossly enlarged with accentuated lobulation and discoloration with the 300 ppm group being more severely effected than the 1000 or 3000 ppm rats. This apparent reversed order of toxicity related to dose could be due to the early mortalities of the high dose rats and, therefore, a short dosing duration. The liver abnormalities seen grossly were associated with increased liver weights and microscopic lesions. Some kidney discoloration and evidence of stomach irritation were also observed grossly at 300 ppm.

In summary, FM-3422 was lethal at doses of 1000, 3000 and 10,000 ppm which is approximately 100, 300 and 1000 mg/kg/day respectively. The liver appeared to be the primary target organ, but there was gross pathological evidence of possible kidney and stomach involvement at the 300 ppm level also.

Study 137-088 - 90 Day Subacute Rhesus Monkey Toxicity Study

FM-3422, suspended in propylene glycol, was administered by gavage to two male and two female monkeys/level using doses of 1, 3, 10 or 30 mg/kg/day. The vehicle appeared to cause anorexia early in the study necessitating volume reduction from 5 to 2 ml/kg. The only mortality occurred in one high dose monkey the last week of dosing. Gastrointestinal signs consisting of emesis, diarrhea and black stools with mucus or bloody mucus were seen in most monkeys from most groups. There were no clinical pathology observations that appeared to be significant compound effects. Pathological lesions reported included lipid depletion of adrenals and atrophy of pancreatic exocrine glands at 30 mg/kg only.

In summary, FM-3422 caused mortality at 30 mg/kg in 1/4 monkeys and appeared to primarily effect the gastrointestinal tract although there was no supporting microscopic evidence.

3/20/79
Date

RAN/lmr

Exhibit 5

Georjean L. Adams
03/29/99 04:22 PM

Corporate Product Responsibility
290-04-01
Tel: 737-4795
Fax: 736-9278

To: David A. Sonstegard/US-Corporate/3M/US@3M-Corporate
cc:
Subject: Re: 8e Follow up - Fish

What I sent to the 8e committee on Friday 1:30pm and Tom's reply:

03 166319

To: Dale L. Bacon/ET-ET&S/3M/US@3M-Corporate
Thomas J. DiPasquale/LA-Legal/3M/US@3M-Corporate
Bill Weppner/US-Corporate/3M/US@3M-Corporate
John P. Pasinski/US-Corporate/3M/US@3M-Corporate
John L. Butenhoff/US-Corporate/3M/US@3M-Corporate
Richard E. Purdy/US-Corporate/3M/US@3M-Corporate
Jeffrey H. Mandel/US-Corporate/3M/US@3M-Corporate
cc:
Subject: 8e Follow up - Fish

ATTORNEY CLIENT PRIVILEGED

03 166320

It has been more than 3 months since we reviewed Rich's hypothesis on food chain contamination. At that time we decided there was insufficient data to support a submission. What is the status of obtaining data to either support or refute the need to report?

---------- Forwarded by Georjean L. Adams/US-Corporate/3M/US on 03/29/99 04:12 PM ----------

Thomas J. DiPasquale
03/26/99 03:21 PM

Office of General Counsel

Building 220-12E-02, 3M Center, St. Paul, MN 55144 USA
Tel: (651) 733-1891
Fax: (651) 736-9469

To: Georjean L. Adams/US-Corporate/3M/US@3M-Corporate
cc: Dale L. Bacon/ET-ET&S/3M/US@3M-Corporate
Bill Weppner/US-Corporate/3M/US@3M-Corporate
John P. Pasinski/US-Corporate/3M/US@3M-Corporate
John L. Butenhoff/US-Corporate/3M/US@3M-Corporate
Richard E. Purdy/US-Corporate/3M/US@3M-Corporate
Jeffrey H. Mandel/US-Corporate/3M/US@3M-Corporate
Subject: Re: 8e Follow up - Fish

Georjean, I'm not sure there is a need to support or refute the hypothesis within any particular time frame. If I recall correctly, the work was itself not part of our formal plan for assessment of environmental

03 166321

Exhibit
1003
State of Minnesota v. 3M Co.,
Court File No. 27-CV-10-28862

3MA01373218

exposure. There are many other theories circulating within the company about paths of exposure, but we cannot undertake extensive efforts to confirm or refute them in each instance. As we discussed earlier this week, a comprehensive exposure assessment plan, with timetables, milestones, objectives, etc. should be our guiding document. This will be needed both for the EPA and for our own purposes. If in the judgment of those who are managing the environmental exposure project the Purdy hypothesis deserves consideration, then it should be incorporated into the comprehensive plan, assigned a priority, and given the necessary resource allocation. I don't see it as standing alone or separate from the broader plan.
Tom

Rich's response to Tom:

Plan! That is the same stalling technique you have been using for the last year. There is a high probability that PFOS is killing marine mammals and you want another plan when we could have had data to support the risk assessment long ago. You were given a plan in 1983. Again in the early 90s. And you authorized no testing.

As I recall we obtained data that eaglets contain PFOS in their plasma last April. Then you as part of an upper management team dispersed the team that initiated the collecting of that data as part of their plan. And then you said we had to put together a plan under the Battelle umbrella. As of now we still have not gotten any data because of that tactic. Battelle is an albatross around our necks and so are you.

Preliminary data indicates that adult eagles have 50 times as much in their plasma than those eaglets. We could have gotten that data and more last summer if we were not stuck planning with Battelle. Don't you realize we have a plan. You continually ignore our plans and start new plans that slows the collection of data essential for our risk assesssments. You slow our progress in understanding the extent of PFOS pollution and damage. For 20 years the division has been stalling the collection of data needed for evaluating the environmental impact of fluorochemicals.

PFOS is the most onerous pollutant since PCB and you want to avoid collecting data that indicates that it is probably worse. I am outrage.

03 166322

Exhibit 6

Com Chem file

Interoffice Correspondence 3M

Subject: MEETING MINUTES -
MEETING WITH H.C. HODGE

3M "CONFIDENTIAL"

JUNE 7, 1979

THOSE PRESENT:

M.T. CASE - 218-2
F.D. GRIFFITH - 220-2E
H.C. HODGE - U. OF CALIFORNIA
L.C. KROGH - 223-6SE
J.D. LAZERTE - 236-1
R.E. OBER - 218-2
J.A. PENDERGRASS - 220-2E
R.A. PROKOP - 236-2B
F.A. UBEL - 220-2E
R.A. Nelson - 218-3

Those present met on April 12, 1979 at the Hilton Hotel in San Francisco California to review recent results which are relevant to the Fluorochemicals in Blood Program and to discuss future plans.

R.A. Prokop began the meeting by giving background on FC-807. FC-807 is used in combination with a hydrocarbon sizing agent to give oil and water repellency to paper and paperboard. One of its principle uses is as an indirect food additive, and a petition was granted in the late 1960's for its use as such. It is manufactured by reacting perfluorooctanesulfonyl fluoride with ethyl amine. Subsequent reaction of the sulfonamide with ethylene carbonate followed by sequential reaction with $POCl_3/H_2O$ and ammonia give FC-807. (See attached flowsheets) It is sold as a 35-40% solution in isopropyl alcohol.

F.A. Ubel reviewed recent developments in the areas of serum organic fluorine levels, human health and epidemiology as they relate to the fluorochemicals in blood program.

The serum organic fluorine level of the individual who was previously removed from fluorochemical exposure when his serum organic fluorine level rose to 70 ppm has recently been measured. The level was found to be 45 + 5 ppm. The amount of FC-143 in his urine was found to be 220 ug for a 24 hour period.

Exhibit
1210
State of Minnesota v. 3M Co.,
Court File No. 27-CV-10-28862

Serum organic fluorine levels have recently been measured on selected employees at Cordova, Decatur and Chemolite. In comparison with levels measured in 1976 and 1977, there has been little change.

The serum organic fluorine levels of 15 Chemolite employees were measured in 1978 prior to their being involved in the packaging of FC-143. Repeat measurements of their serum organic fluorine taken recently showed some unexpected elevations in levels. Inorganic levels were also higher than expected. Contamination with inorganic fluoride is suspected to be the cause of the high inorganic levels in serum, but the entire set of results will be reviewed to see if any errors were made to relate exposure to serum organic fluorine levels.

Eight serum samples from rural China were analyzed for organic and inorganic fluorine levels. Organic fluorine levels ranged from 0.004 ppm to 0.017 ppm and inorganic fluoride levels from 0.044 to 0.076 ppm. In the U.S. serum organic fluorine levels range from 0.002 to 0.13 ppm and inorganic levels from 0.003 to 0.17 ppm.

The epidemiology study is still in progress. The study involves tracing about 3500 people and involves about 100 deaths. So far there does not appear to be what might be considered as "unusual causes of death".

H.C. Hodge commented that data from this epidemology study is very important and that the study should be carried out carefully. Criteria set forth by the N.C.I. should be followed. Dr. Hodge further stated that he would rather accept data from this type of study for identifying human health effects than data from animal studies, but that the National Cancer Institute would not agree.

F.A. Ubel summarized a conversation with two Mayo Clinic physicians regarding the significance of slightly elevated liver enzyme levels in some of the Chemolite employees. No definite conclusions were reached, except that the values were very slightly elevated and in some instances were felt to be compatible with their history of alcohol ingestion. They could not make any statement unless they had a chance to examine the employees personally.

Dr. Hodge was shown a summary of the abnormal findings in those 3M employees who had known fluorochemical blood levels. He was given a copy.

The results of the physical examinations done on Decatur and Cordova employees look very good. There does not appear to be evidence of a work related problem. The Chemolite employees showed more abnormalities, but the majority of these appeared to be related to a known medical problem or medication. It was the conclusion of the physicians who supervised the examinations that there did not appear to be a problem which could be identified as work related. Additional analysis of the data will be done.

J.A. Pendergrass reviewed data on workplace concentrations of various fluorochemicals in Alabama and Minnesota plants. In most cases the workplace concentrations of fluorochemicals are low-lower than some time weight averages recommended for known or suspected carcinogens. Fluorochemical salts are exceptions. Due to their dusty nature workplace concentrations are higher. Levels of one of these materials, FC-143, have been reduced to an acceptable value. Work is underway to reduce exposure to other fluorochemical salts.

R.A. Nelson reviewed results of 90 day subacute toxicity studies using FC-95, FC-143 and FM-3422. (Slides attached) Of these compounds FC-95 was the most toxic. It produced deaths in the monkey at 4.5 mg/Kg and in the rat at ~10 mg/Kg. Target organs in the rat were liver hematopoietic tissue, stomach and small intestine. In monkeys, the apparent target organ was the upper G.I. tract.

The liver and possibly the kidney and G.I. tract were target organs in the rats fed FM-3422. In the case of monkeys there was clinical evidence of G.I. toxicity.

FC-143 produced liver changes in rats at the highest dose. In monkeys hematopoietic tissue was affected at lethal doses and there was evidence of gastrointestinal toxicity. H.C. Hodge also presented his summary of results of the 90 day studies on FC-143, FM-3422 and FC-95 (attached). His projected no-effect levels were 1.5 mg/Kg for FC-143 and FM-3422 in the rat, and 3 mg/Kg in the monkey. For FC-95 there was no no-effect level in the rat and no data for estimating a no-effect level in the monkey. There appears to be an indication of liver effects from FC-95, FM-3422 and FC-143 at all dose levels in the rat studies.

H.C. Hodge questioned whether some of the toxic effects observed in the animal studies might be due to low surface tension. Surface active agents are known to be capable of causing a problem in the gut. It was pointed out that some fluorochemicals are the most potent surfactants known.

F.A. Griffith pointed out that toxic effect such as those observed with FC-95, FM-3422 and FC-143 are common with surfactants.

R.A. Nelson pointed out that the liver effects are probably reversible, but this would require further study to prove.

H.C. Hodge pointed out that a buildup of fluoride ion can be detected most rapidly in the bone. Thus evidence of breakdown of fluorochemicals to fluoride ion could be detected in the bone. Bone should be collected at the end of a chronic study and analyzed for fluoride.

H.C. Hodge was then asked for his recommendations on future work. After a brief review of customer exposure to 3M fluorochemicals Dr. Hodge recommended the following:

1. It does not appear that there is a toxicity problem outside 3M. However we do not know if there are toxicity problems due to employee exposure. Reduction in exposure should have top priority.

2. Metabolic instead of chronic studies should be done next. Sequestering kinetics and metabolites should be looked at.

3. Reproduction studies, including teratology, should be given high priority. Two generations should be used. Such studies should take less than one year.

4. Carcinogenicity of fluorochemicals should be looked at. Start with Ames testing and continue to more sophisticated mutagenicity tests. If any of these turn out positive it will be necessary to go on to a chronic study.

R.A. Nelson questioned the reliability of tests other than the Ames. The transformation test may be on the way out. Different regulatory agencies are not getting results which agree.

M.T. Case outlined proposed studies on FC-807 and studies being considered for FC-95 and FC-143. (Slides attached) A question was raised on the FC-807 study as to whether the F.D.A. should be aksed about the protocol. It was suggested that we should proceed without consulting the F.D.A. since the study is scientifically valid.

R.E. Ober outlined planned work in the metabolic area. (Slides attached) This proposed work involved metabolism of FC-807, persistence of FC-95 and FC-143, and skin absorption studies.

It was suggested that H.C. Hodge be given more time to consider the proposed slides on FC-807, FC-95, FC-143 and the metabolism work before being asked for an opinion.

ADDENDUM

I called Dr. Hodge on April 20, 1979 to give him the acute oral toxicity data on FC-95, FC-143 and FM-3422 which was generated at IRDC prior to the 90 day studies. He asked that the following be added to the meeting minutes:

The study of levels of FC-807 or its metabolites is of utmost importance in determining possible future problems. It should be determined if FC-807 or its metabolites are present in man, what level they are present, and the degree of persistence (half-life) of these materials.

R.A. Pribyl

RAP/ko
Attachments

Exhibit 7

Return to FM 3422 File

Form 6747-11-A

TECHNICAL REPORT SUMMARY

Date: 5/22/79

TO: TECHNICAL COMMUNICATIONS CENTER – 201-2CN

(Important – If report is printed on both sides of paper, send two copies to TCC.)

Field	Value
Division	Environmental Laboratory (EE & PC)
Dept. Number	0222
Project	Decatur, Alabama - Tennessee River Fish
Project Number	78-2740
Report Title	Bioaccumulation of Fluorochemicals in Tenn. River Fish
Report Number	001
To	D. L. Bacon
Author(s)	James E. Gagnon
Employee Number(s)	213531
Notebook Reference	51568
No. of Pages Including Coversheet	10

SECURITY ▶ ☐ Open (Company Confidential) ☒ Closed (Special Authorization) | 3M CHEMICAL REGISTRY ▶ | New Chemicals Reported ☐ Yes ☒ No

KEYWORDS: (Select terms from 3M Thesaurus. Suggest other applicable terms.)

EE & PC
Decatur

Should be F 6309

Everything mentioned in report according to JEG on 7-22-81

CURRENT OBJECTIVE: Qualitative and quantitative determination of ~~FM-3923~~, FM-3925, and FM-3422 in fish taken from the Tennessee River above and below Wheeler Dam at 3M's Decatur plant. Analyze for organic and inorganic fluoride in the same samples.

REPORT ABSTRACT: (200-250 words) This abstract information is distributed by the Technical Communications Center to alert 3M'ers to Company R&D. It is Company confidential material.

Channel catfish *(Ictalurus punctatus)* had the largest combined total, 2.74 ppm and 1.13 ppm, of FM-3923, FM-3925, and FM-3422, as determined by gas chromatography. It was shown that the three fluorochemicals of interest bioaccumulated more readily in the gastrointestinal tract, fat and reproductive system of the channel catfish, while no fluorochemicals were observed in the muscle layer. A white bass *(Roccus chrysops)*, taken from below Wheeler Dam, had a combined FM-3923, FM-3925, and FM-3422 concentration of 0.40 ppm. A white crappie *(Pomoxis annularis)*, from above Wheeler Dam, was found to contain only FM-3923, 0.004 ppm.

Total organic fluoride ranged from 9.7 ppm, channel catfish, to 16.2 ppm, white crappie. Inorganic fluoride ranged from 6.2 ppm, white bass, to 24.6 ppm, channel catfish.

Future Studies: TLC on above fish samples.
GC/MS on channel catfish samples.
Background fluorochemical analysis on a crappie from a Minnesota Lake.

cc: D.Ricker-236-2B
A.Welter
A.Mendel

Information Liaison Initials: SKW

3M CONFIDENTIAL

Exhibit 1208
State of Minnesota v. 3M Co., Court File No. 27-CV-10-28862

3MA01409559

INTRODUCTION

It is known that 3M's Decatur, Alabama plant effluent has high organic fluoride levels, 10.9 ppm (1)(2). It has also been shown that fluorochemicals can bioaccumulate in fish in a laboratory environment (3)(4). With these combined factors, the next step was to see if fish caught in the Tennessee River near the Decatur plant had detectable levels of fluorochemicals.

RESULTS AND DISCUSSION

Table 1 lists the concentration, in ppm, in fish of compounds which have the same retention time as the three fluorochemicals of interest (FM-3923, FM-3925, and FM-3422).

Analysis of the results for the dissected channel catfish, Sample 3A, shows that the fluorochemicals bioconcentrate to a greater extent in the gastrointestinal tract, reproductive system, and fat. It can also be seen that the muscle layer was found not to bioaccumulate the three fluorochemicals of interest. These results agree with earlier reports (3)(4).

When comparing the total fluorochemical content (TFC) for the two whole fish samples, the larger channel catfish contained more than twice the fluorochemical content, 2.74 ppm vs. 1.13 ppm. Since both fish were caught in the same area, a reasonable explanation for this may be related to the high partition coefficients for channel catfish. Fluorochemicals bioaccumulate in fatty tissue, and since more fatty tissue is present in the larger fish, more fluorochemicals would be expected.

FM-3923 is present at higher concentrations in the dissected channel catfish, sample 3A, than other samples. Since bioaccumulation rates have not been determined for FM-3923, no explanations for the higher concentrations can be offered.

The two fish samples which had cores taken from them will not be rigorously compared to whole fish samples. The reason for this is that the core samples may not have representative concentrations of fluorochemicals (whole fish values may be higher or lower). Since core samples were taken from the approximate same location, the results can be rigorously compared.

The white bass from below Wheeler Dam, sample 1B, had a whole fish TFC of 0.40 ppm, while the white crappie from above Wheeler Dam, sample 2A, had a whole fish TFC of 0.004 ppm. With such small statistical samples, it would be difficult to say that the larger TFC is due only to the white bass living in the presence of higher fluorochemical concentration, downstream from the plant. Other possible explanations for the higher TFC could be the following:

TABLE 1

FLUOROCHEMICAL CONCENTRATION (ppm)
IN TENNESSEE RIVER FISH

Sample	FM-3923	FM-3925 & FM-3422 (1)	Total Combined FC in Fish (ppm) (2)
1A - Whole fish	0.40	0.73	1.13
1B - Core (3)	0.82	3.31	0.40 (4)
2A - Core (5)	0.06	N.D. (6)	0.004 (4)
3A - Gills	1.48	0.80	
3A - Liver	2.17	0.38	
3A - Parts (7)	1.33	0.43	
3A - Muscle	N.D.	N.D.	2.74 (9)
3A - Fat (8)	13.85	6.12	
3A - Gall bladder	1.57	0.74	
Water blank	N.D.	N.D.	
Ethyl acetate blank	N.D.	N.D.	

Footnotes to Table 1:

(1) FM-3925 and FM-3422 cannot be resolved with GC parameters used; therefore, a combined value is reported.
(2) Based on frozen weight of the fish.
(3) Sample core, 3.61 cm, id contained skin, filet, reproductive organs, and parts of kidney, rectum, and backbone.
(4) Assumes that the concentrations obtained in the core are representative of the rest of the fish.
(5) Sample core, 3.61 cm id contained filet, vertebrae, skin, and bile.
(6) N.D. = Not detected.
(7) Consisted of muscle, skin, blood, bone, and cartilage.
(8) Consisted of gastrointestinal tract, reproductive system, and fat.
(9) Based on the actual weight of sample used, 18.8% less than frozen weight, and weight percent of each part.

1. Longer river residence time, older fish.
2. Longer location residence time.
3. Different species
   a) Different feeding and life stylos
   b) Contains larger weight percent of organs which tend to bioaccumulate fluorochemicals
   c) Larger fluorochemical partition coefficients

If the core samples are representative of whole fish concentrations, then it can be postulated that channel catfish bioaccumulate fluorochemicals to a greater extent than either white bass or crappie. Reasons for this are the same as listed above.

Table 2 gives the results of the organic (RF) and inorganic fluoride (F^{θ}) concentration, in ppm, in the fish samples.

TABLE 2 (5)

ORGANIC (RF) AND INORGANIC (F^{θ}) FLUORIDE CONCENTRATIONS (ppm)

Sample	RF	F^{θ}
1A	9.7	24.6
2A	16.2	13.3
1B	10.5	6.2
Water	N.D.	0.01

Jon Belisle points out that the high inorganic fluoride values seem rather surprising. His only explanation was that fish flour previously analyzed, for a different requestor, was shown to have inorganic fluoride values higher than organic fluoride. Jon also states that high inorganic fluoride values would make it difficult to calculate low levels of organic fluoride.

Comparison of the organic and inorganic fluoride content shows that samples from above Wheeler Dam have just as high, if not higher, values than for the sample from below the dam. There are no clear cut explanations for this observation. An earlier analysis of Tennessee River water showed high organic fluoride concentrations upstream from the plant. At that time, it was thought the samples may have been mislabeled. With these results,

it would seem to indicate that the concentration of fluorochemicals may actually be less below Wheeler Dam. This may be caused by volatilization of the fluorochemical whon going over thc dam (1), settling of fluorochemicals before the dam.

Comparison of organic fluoride values from Tables 1 and 2 show no correlation. For example, the highest organic fluoride value, 16.2 ppm for sample 2A, had the lowest TFC, 0.004 ppm, for the fluorochemicals analyzed. A possible explanation is that there are organic fluorides present in very high concentrations which were not analyzed for individually. The species which had the highest fat content, channel catfish, had the lowest organic fluoride concentrations.

With limited sample population (2 fish of one species and one of each of two other species), it is difficult to draw any meaningful conclusions. The only definite conclusion is that the fluorochemicals studied do appear to bioaccumulate in river fish under natural conditions.

EXPERIMENTAL

1. Sample materials

Fish

1A - Small channel catfish *(Ictalurus punctatus)*, caught above Wheeler Dam in Tennessee River.

1B - White bass *(Roccus chrysops)*, caught below Wheeler Dam in Tennessee River.

2A - White crappie *(Pomoxis annularis)*, caught above Wheeler Dam in Tennessee River.

3A - Large channel catfish *(Ictalurus punctatus)*, caught above Wheeler Dam in Tennessee River.

Standards

FM-3923, FM-3924, and FM-3422.

Ten ppm standards of FM-3923, FM-3925, and FM-3422 were prepared by diluting 1 ml of a 100 ppm standard, in ethyl acetate, to mark with ethyl acetate in separate 10 ml volumetric flasks.

2. Analysis Instruments/Materials

Blender:

Waring Commercial blender, Model #91-263, available from Waring Products Division, Route 44, New Hartford, CT 06057.

Tissuemizer:

Model #SDT, available from Tekmar Company, P. O. Box 37202, Cincinnati, OH 45222.

Dinker Die:

3.61 cm id AISI-02 high carbon steel cutting die made by Jerry Guthrie in Central Research Labs, described in 3M Technical Notebook #51568-35.

Mixer:

"Vortex Genie" Model #K-550-G, available from Scientific Industries, Inc., Bohemia, NY 11716.

Centrifuge:

Damon-IEC Model #B-20A, available from Damon-IEC Corporation, Needham Heights, MA.

Bottles:

Four-ounce widemouthed clear glass bottle sealed with aluminum foil and aluminum foil-lined caps.

125-ml linear polyethylene (LPE) plastic bottle with polyseal caps.

Gas Chromatograph:

Chromatograph - Hewlett-Packard Model 5713 GC.
Integrator - Hewlett-Packard Model 3380A integrator-printer.

Both of the above available from Hewlett-Packard Co., 150 Page Mill Road, Palo Alto, CA 94304.

Column - Six-foot, 1/8 inch OD, stainless steel, packed with 10% CW20M on 60/80 Chromasorb W-AW.

Column Temperature - Isothermal 180° C.
Injector - On-column at 200° C.
Detector - Electron Capture at 300° C.
Flow - ∿40 cc/minute of Argon:Methane (95/5).

Ethyl Acetate:

"Li Chrosolv" chromatography solvent available from MC/B Manufacturing Chemists, 2909 Highland Avenue, Norwood, OH 45212, as Catalog #6008688M.

Water:

Deionized water.

3. Procedure (6)

Procedures used below, except for minor modifications, were obtained from earlier 3M Technical Report summaries (7).

Samples 1A through 3A and 1B were removed from the freezer and placed in large aluminum pans, in a fume hood, and allowed to thaw.

A whole channel catfish, sample 1A, was cut into 5 sections and homogenized in a blender with 200 ml water.

Sample 1B had a dinker die core sample taken just off the lateral line behind the gill plate. Contents of the 20.591 gram sample were skin, filet, small part of backbone, reproductive organs, part of kidney, and rectum.

Sample 2A had a dinker die core sample taken behind the gill plate. The 16.684 gram sample contained filet, vertebrae, skin, and bile. Samples 1B and 2A were homogenized with 10 ml of water in a "tissuemizer."

Sample 3A was dissected, and the various individual parts were homogenized with water. Individual parts weighing more than 25.0 grams were homogenized in a blender, while those of lesser weight were homogenized in a "tissuemizer." Table 3 lists the sample, sample weight, and amount of water added for homogenizing each sample.

All of the above samples, after homogenization, were divided into five aliquots and placed in precleaned bottles, (dichromate/acid, water rinse, dry, toluene, dry). Three aliquots were placed in LPE bottles, while the other two were placed in glass bottles. Samples were stored in a refrigerator at 4.5° C. until needed.

Samples analyzed for FM-3923, FM-3925, and FM-3422 were prepared according to the following procedure. See Table 4 for weight of sample and milliliters of ethyl acetate used for extractions.

A previously homogenized sample, stored in a glass bottle, was weighed (no larger than 4.00 g) and added to a 30-ml precleaned glass centrifuge tube. A volume of ethyl acetate was added at the rate of 1.0 ml ethyl acetate per gram of homogenate. The ethyl acetate/fish homogenate were mixed for 1.5 minutes in a mixer at a speed setting of 3. The samples were removed and centrifuged at 1500 rpm at

21° C. for 10 minutes. After centrifuging, the ethyl acetate layer was separated, by use of a pipet, and placed in a vial. Five µl of sample (standard) was injected for gas chromatographic analysis.

Samples 1A, 2A, and 1B homogenates, plus a water blank, in LPE bottles, were sent to Jon Belisle of the Central Research Laboratory for organic and inorganic fluoride analysis.

REFERENCES

(1) 3M Technical Report Summary, August 30, 1978, Arthur Mendel to R. L. Bohon, "Fate of Fluorochemicals Project - Progress Report."

(2) Central Research Laboratory Report Number 6902, April 20, 1978, Jon Belisle.

(3) "Bioconcentration of FM-3422 in Bluegill Sunfish and in Channel Catfish," M. T. Elnabarawy to A. N. Welter, May 17, 1977.

(4) 3M TRS, August 16, 1978, A. N. Welter to D. L. Bacon, "Evaluation of the Bioconcentration Potential of FM-3422."

(5) Central Research Laboratory Report on Request #A72199 by Jon Belisle, May 7, 1979.

(6) Experimental work done in cooperation with A. N. Welter of the Environmental Laboratory (EE & PC), who performed the dissections and homogenizations.

(7) 3M Technical Report Summary, November 15, 1977, A. Mendel to D. L. Bacon, "Analytical Methodology on FM-3422."

James E. Lagnon
JEG/cen

TABLE 3

FISH WEIGHTS AND WATER VOLUMES USED FOR HOMOGENIZATION

Sample Description	Initial Whole Frozen Weight	Actual Sample Weight Used	ml Water Used
1A	146.0 g	Whole fish (1)(2)	200
2A	266.5 g	16.684 g (3)	10
1B	210.0 g	20.591 g (3)	10
3A - Muscle	752.0 g	209.93 g	200
3A - Gall bladder	752.0 g	1.378 g	10
3A - Liver	752.0 g	5.949 g	10
3A - Fat	752.0 g	52.230 g	100
3A - Parts	752.0 g	321.57 g	300
3A - Gills	752.0 g	19.38 g	100

Footnotes:

(1) A fish hook, with no apparent rust or line, was found in fish and was removed before homogenization.

(2) The fish appeared to be slightly dehydrated (possibly due to constant air flow over surface of fish) so the actual weight of fish used may have been less than frozen weight.

(3) Sample core 3.61 cm id.

TABLE 4

FISH WEIGHTS AND ETHYL ACETATE VOLUMES USED FOR EXTRACTIONS

Sample Description	Weight of Fish Homogenate (grams)	% Water in Homogenate	Actual Fish Wt. Extracted (mg)	ml EtOAc
3A - Gall Bladder	1.20	87.9	145.2	1.2
3A - Liver	2.20	62.7	820.6	2.2
3A - Muscle	2.40	48.8	1228.8	2.4
3A - Fat	2.40	65.7	823.2	2.4
3A - Parts	3.00	48.3	1551.0	3.0
3A - Gills	3.00	83.8	486.0	3.0
Water Blank	2.40	100.0	--	2.4
1A	2.40	57.8	1012.8	2.4
1B	2.40	32.7	1615.2	2.4
2A	2.40	37.5	1500.0	2.4

Exhibit 8

3M | RIKER LABORATORIES, INC.

Interoffice Correspondence:

July 6, 1979

cc: F. D. Griffith

TO: R. A. Nelson

FROM: M. T. Case

SUBJECT: Fluorochemical Chronic Toxicity

In my response to Drs. Hodge and Mitchell recommendations, I strongly recommended that a chronic rat study on a fluorochemical be started as soon as possible. At that time FC-807 was indicated as my choice but in view of the fact that given the very low potential human exposure via an indirect food additive the FDA would not ask for carcinogenicity data, the decision not to proceed with the FC-807 study is understandable. As I sat listening, several weeks ago, to both John Favorite and Tom Scheuerman talk about being responsible 3M scientists in the area of toxicity testing, I was reminded of the lack of chronic toxicity data on 3M fluorochemicals.

I believe it is paramount to begin now an assessment of the potential (if any) of long term (carcinogenic) effects for these compounds which are known to persist for a long time in the body and thereby give long term chronic exposure. Over a year has passed since the completion of the IRDC 90-day rangefinder rat studies and to delay chronic testing any longer is not warranted. As I indicated before, I think that both the metabolic and chronic toxicity studies are important and must go forward. However, metabolic studies will not provide answers in the area of potential of any long term (carcinogenic) effects. In my opinion, 3M should have carcinogenicity data on at least two of its fluorochemicals and chronic rat toxicity studies need to be started soon - one yet in 1979 and the other in 1980. The 1979 study could be on FC-143 (we already have rangefinder information) - say at dose levels of 300, 100 and 10 ppm in the diet.

M T Case

M. T. Case

MTC/sl

Exhibit
1212
State of Minnesota v. 3M Co.,
Court File No. 27-CV-10-28862

OFS-128

3MA00593079

Exhibit 9

AD-776 205

A SUBSTITUTE LIQUID FOR AFFF (AQUEOUS FILM FORMING FOAM) CONCENTRATE FOR CHECKING PROPORTIONERS

R. L. Gipe, et al

Naval Research Laboratory
Washington, D. C.

February 1974

UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

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	6. PERFORMING ORG. REPORT NUMBER
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Fire Suppression
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Concentrate

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

The high cost of Aqueous Film Forming Foam (AFFF) concentrate makes it expensive to conduct the routine testing of shipboard High Capacity Fog Foam station proportioners and has created the desire for a lower cost substitute material. The selection of an appropriate liquid to simulate the performance of an AFFF concentrate in a proportioning system is based on matching the viscosity characteristics for pumping and the index of refraction characteristics for analysis.

(Abstract continues)

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20.

Mixtures of glycerin and water were found capable of reproducing these properties of all existing AFFF concentrates when used in strengths of 40 to 87% glycerin. The extra logistics and manipulations required to use a simulated liquid should be considered in making a decision on its use.

CONTENTS

A SUBSTITUTE LIQUID FOR AFFF CONCENTRATE FOR CHECKING PROPORTIONERS

R. L. Gipe, C. S. Butler and H. B. Peterson

BACKGROUND

The routine maintenance procedures for the High Capacity Fog Foam stations aboard aircraft carriers and some systems on other vessels require that a check be made on the operation of the water-motor proportioners. One level of checking can be accomplished by recirculating AFFF concentrate through the proportioner pump and back into the tank; however, the highest level of checking can only be done by actually pumping concentrate into the risers supplying the outlets. A serious drawback to the more frequent checking of the stations has been the high cost of the AFFF concentrate which is necessarily consumed. It has been proposed that a material simulating the characteristics of AFFF concentrate, but available at a lower cost, would be of benefit to the Navy and NRL was requested to recommend a suitable substitute.

INTRODUCTION

From the work done over the past years it has been found that viscosity is one of the more significant properties of the liquid being handled in the positive displacement pump portion of the proportioner. The high pressure existing on the discharge side of the pump forces liquid back through the axial and radial clearances in the pump to the low pressure existing at the intake side. The more viscous the liquid, the less the volume of liquid passing through these narrow openings.

The objective was thus to find a method of duplicating AFFF concentrate viscosity at a reasonable cost. It was also judged to be of importance to have a material with a suitable refractive

Manuscript submitted January 14, 1974.

index range so that the solutions could be analyzed by the same hand refractometer presently used for AFFF solutions.

High molecular weight polymers of ethylene oxide and carboxymethylcellulose are dry materials which, added to water in small amounts, will increase its viscosity sufficiently, but both were judged to be too difficult to handle and put into solution. Glycerin, a readily available liquid material, was also tested for use in this investigation and was found to give the desired performance. Because of its low cost, ready availability and appropriate refractive index, other potential agents were not investigated.

EXPERIMENTAL RESULTS

The first AFFF concentrate fully compatible with sea water and used in shipboard systems was designated FC-195. This formulation has been followed by others known as FC-196, FC-199, and FC-200. A considerable drop in concentrate viscosity occurred in those materials subsequent to FC-195. The viscosity-temperature characteristics of each of these concentrates are given in Figure 1. Data are also given for protein foam liquid, for which the proportioning equipment was originally designed, for FC-200 Lot 3002, an experimental concentrate made to have a viscosity comparable to protein foam liquid, and for FC-194, an earlier fresh water compatible material.

All the viscosity measurements reported in this work were determined with a capillary viscometer of the appropriate size and in accordance with ASTM D445-65.

Figure 2 is a plot of the viscosities of mixtures of water and glycerin and shows the range of viscosities that can be achieved with this system at 77°F.

A comparison was made between the temperature-viscosity characteristics of a water-glycerin mixture and an AFFF concentrate. These data are given in Figure 3.

Finally, a series of runs was conducted on a FP-1000 proportioner comparing actual AFFF concentrates and glycerin-water mixtures of similar viscosities in order to detect the existence of any non-viscosity related phenomena. (The viscosities of the liquids were selected in part to check the characteristics of the

proportioner over a small range of concentrate viscosities.) Five liquids were employed: (1) water with a viscosity of 1 centistoke (CS), (2) an AFFF concentrate diluted with water to give 3 CS, (3) a glycerin-water mixture of 3 CS viscosity, (4) a mixture of AFFF concentrates to give 6 CS, and (5) a glycerin-water mixture of 6 CS viscosity. These results are summarized in Figure 4.

Refractive index measurements were made of the water-glycerin mixtures to see if the optical properties of the solutions were sufficient to provide a workable range on the hand refractometer now used on shipboard for AFFF solution analysis. In Figure 5 are plotted a concentration-refractive index relationship for an AFFF concentrate and a corresponding curve for a simulated concentrate made up of glycerin and water. The glycerin-water mixture selected was of such a ratio as to create a 3 CS viscosity concentrate. This meant that the glycerin content was very low and would represent the lowest amount which would ever be used in a simulated concentrate.

DISCUSSION

Performance of Glycerin-Water Mixtures

From the data of Figure 1 it can be observed that there have been considerable changes in the viscosity of AFFF concentrates over the years of their development. None of them has been very close to the characteristics of protein foam liquid for which most of the fire fighting equipment in the Navy was designed. In those systems where the concentrate proportioning is viscosity dependent, the delivered concentrations will have been affected accordingly. In many cases the "reserve strength", or safety factor, inherent in the concentrate composition has meant there has been little compromise in fire suppression capability with either lean or rich solutions. However, in some extreme cases it could have meant the complete lack of concentrate introduction. An earlier report (1) has covered this subject in detail.

The characteristics of Lot 3002 indicate that it is not a problem to reproduce a viscosity comparable to protein foam with an AFFF concentrate. The most probable explanation for why it has not been done earlier has been the lack of appreciation of the shipboard proportioning requirements and the limited amount of concentrate used on shipboard, as compared to other consumers.

The range of viscosities, Figure 2, which may be obtained by mixtures of glycerin and water varies from 1 CS for pure water to approximately 900 CS for pure glycerin at 77°F. This range easily encompasses the viscosities of all of the concentrates covered in Figure 1 and at all temperatures between 32° and 120°F. Thus, glycerin-water mixtures have no limitations in this area and the curve in Figure 2 will enable selection of the proper proportions to simulate any AFFF or protein concentrate.

The effect of temperature change on the viscosity of a glycerin-water mixture is given in Figure 3 and is compared to an AFFF concentrate of approximately the same viscosity. This was done in order to determine if an error would be introduced if a glycerin-water mixture was selected to match an AFFF concentrate at 77°F but then actually used at a different temperature. Examination of the two functions show that they are not precisely parallel, indicating their temperature characteristics are not identical. However, it is believed that no appreciable error would be introduced. A glycerin-water combination prepared to the same viscosity as FC-199 at 80°F would have close to the same viscosity as FC-199 if both were taken at 40°, or to 100°F, and any results obtained with glycerin-water should be acceptable.

Figure 4 gives the actual FP-1000 water-motor proportioner performance comparison between AFFF concentrate and a glycerin-water simulated concentrate at two viscosities, 3 and 6 CS. Although there was some difference noted between the two liquids at 3CS, there was essentially no difference at 6 CS and it is believed that the glycerin-water mixtures may be taken as fully acceptable substitutes for the concentrates. It may also be seen from these data that the solution concentrations were noticeably affected by even slight changes in the viscosity of the material being pumped.

During the period of testing when AFFF concentrate was being recirculated as a conservation practice, the viscosity was checked regularly in order to ascertain whether the shearing action of the pump was affecting a breakdown of any polymers in the fluid which might influence the viscosity. No evidence of shear degradation was detected.

The data in Figure 5 show the relationships of concentration and refractive indices for glycerin-water and AFFF solutions. The results for AFFF are similar to the calibration curves prepared on shipboard preparatory to making an operational check of a system. Normally such a calibration curve is made up for each station as it is checked because the different concentrates produce different slopes, and sea water contamination, if present, will shift the curve downward and introduce errors in the final analyses. The glycerin-water mixture shown in Figure 5 was chosen to simulate a 3 CS concentrate. Because the glycerin-water mixture curve and the AFFF curve both fall well within the range of the hand refractometers now in use, no analytical problem would be encountered by the use of glycerin-water mixtures as a substitute concentrate. On the contrary, the increased steepness noted in the glycerin-water mixture will increase the accuracy of determinations over the AFFF concentrates.

Estimated Cost

The glycerin used in the study was made by Shell Chemical Co., 99.5% purity, and cost $0.29 per pound, or $3.05 per gallon, purchased in a small quantity from Baltimore Chemical. This cost per gallon probably represents the highest possible for this grade, and purchase in commercial quantities would reduce the price to approximately $0.24 per pound.

The cost per gallon of simulated AFFF concentrate will, of course, depend on the viscosity of the specific concentrate to be simulated. Table I presents the estimated costs of the glycerin (based on the NRL cost) required to make up one gallon of simulated concentrate. The balance of the mixture is water.

Table I

Glycerin Costs for Simulated AFFF Concentrates

Concentrate	Glycerin Content %	Glycerin Cost Per Gallon Mix	Viscosity 80°F Centistokes
FC-195	87	$2.70	110.
FC-196	50	1.52	6.1
FC-199	48	1.47	5.9
FC-200	40	1.22	4.2
Protein	70	2.14	20.

With the current costs of FC-200 running about $5 per gallon, it may be seen that the simulated version of FC-200 would cost about one quarter that of the real material.

Operational Considerations

Although the substitution of a glycerin-water mixture would reduce the cost of material consumed in running a proportioner check, the operational considerations must also be taken into account.

Emptying of the 600-gal. storage tanks, replacing with a simulated concentrate, running a check, and then putting the original concentrate back into the tank would appear to be an almost prohibitive procedure. The alternative would be to install a tee fitting in the proportioner suction line downstream of the Powertrol valve which would permit connecting a 50-gallon tank of simulated material brought to the station for the purpose. In addition to the costs of modifying the piping arrangement at each station, new possibilities for errors will be introduced and reliability will be compromised.

A test procedure would have to be worked out around the objective of the test. If the objective was to ascertain what a particular proportioning station would do under operational conditions, the viscosity of the contents of the tank would have to be determined and the simulated concentrate prepared accordingly for that station. This would be a fairly sophisticated operation for shipboard personnel to conduct. Tanks on aircraft carriers have been found to contain materials ranging in viscosity from 4 CS to 100 CS. If the objective was to ascertain the relative operating condition of a proportioner, a standard mix of simulated concentrate could be used for all stations.

By the use of appropriate tables, data obtained with a 6 CS simulated concentrate could be translated into what the solution strength would have been for the actual viscosity of the concentrate in the tank. Also, the minimum acceptable limit for proportioner performance could be established on the basis of a certain fixed viscosity. For example, every proportioner to be classed as acceptable would have to achieve an output solution strength of at least 4.5% when using a test concentrate of 6 CS at a stipulated flow rate. (In those cases where the tank contents were of a higher than 6 CS viscosity, higher percentages would be required

at the low end of the flow scale in order for the unit to be considered acceptable.

Environmental Impact

The possibility of conducting proportioner tests while the ship is in port raises questions as to the environmental considerations involved. These might be different from those when testing at sea.

The Director of the Advisory Center on Toxicology of the National Research Council/National Academy of Sciences has stated (2) that solutions of glycerin in water are biodegradable in the sea. The 3M Co. has also stated that its AFFF product is biodegradable and will have no adverse effects on the environment (3). The above statements would indicate that either product could be used at sea or in port without creating a problem, however, such might not be the case. Under the strictest interpretations, practically anything undrinkable by humans is unfit to discharge over the side into the sea or into an estuary. Whether a concentrate, or substitute concentrate, is simply biodegradable may not be adequate justification for its use.

Certainly visibility of any discharge over the side is an important aspect and a large raft of snow-white AFFF floating in a harbor is definitely an attention-getter. Aeration of a glycerin-water mixture does produce a froth but it is less stable than AFFF. From this standpoint, perhaps either material might be unacceptable for in-port discharge. In the event the visibility problem does become the critical one, other test techniques could be worked out whereby a non-aerated discharge could be run through a closed hose system over the side, but terminating below the water surface or into a shore sanitary system.

Other Methods

After the above work with glycerin had been completed, the 3M Company reported on some similar work they had done using polyvinyl alcohol as a thickening agent. They prepared a concentrate with a duPont product, Elvanol 71-30, in a 35% ethylene glycol-water mixture. Further dilution in water gave liquids of 6, 15, and 30 CS viscosity with 1, 2, and 3% respectively of Elvanol. The ethylene glycol was added in order to provide for concentration analysis by means of the refractometer. According

to 3M this is also a suitable substitute.

3M also prepared a concentrate of high viscosity, 9000 CS, which could be used to increase the viscosity of FC-196, FC-199, and FC-200 already in shipboard tanks without decreasing their fire performance capability. Typically, adding 10% by volume would raise the viscosity from 4 to 20 CS.

Such an approach could be taken to prepare a simulated concentrate from water on shipboard. (It would not be necessary, however, to add the fluorocarbon components.) Slightly over 15% of this concentrate would be required to increase the viscosity of water from one to 20 CS. However, effort is required to dissolve this concentrate uniformly in water.

The 3M Company is presently giving consideration to the marketing of such a product specifically for the testing of fire suppression systems. Apparently there has also been an expression of interest from the owners of industrial installations. No information is available as to a proposed cost of the material.

CONCLUSIONS

It is feasible to simulate AFFF concentrates for proportioner testing by adding appropriate agents to water to give it the proper viscosity and refractive index.

The cost of simulated concentrates will depend in part on which AFFF concentrate it is desired to simulate because their viscosities vary. The cost will also depend on whether it is procured as a ready-to-use mixture or whether the final preparation and mixing is done on-site from a highly concentrated liquid. However, the final costs could be on the order of half the present cost of real AFFF concentrate.

NRL has found glycerin-water mixtures to be suited for the purpose and the 3M Company has found polyvinyl alcohol-ethylene glycol-water mixtures to be suitable also.

RECOMMENDATIONS

The use of simulated concentrates for proportioner testing on shipboard is not recommended unless no other recourse is available. It is believed that the logistical problem of having a simulated concentrate in the supply system, the operation of change-over from real concentrate to simulant and then back to real concentrate for each test, and the increased potential for introducing errors and confusion would not be justified on the basis of the differential costs per gallon of the simulated and real concentrates.

In the event that it is decided for ecological reasons to proceed with the use of a simulated concentrate in spite of the cited problems, it is recommended that commercial sources be approached as potential suppliers of a simulated AFFF concentrate based on a glycerin-water mixture or a polyvinyl alcohol-ethylene glycol-water mixture.

REFERENCES

1. Gipe, R. L. and Peterson, H. B., "Proportioning Characteristics of Aqueous Film-Forming Foam Concentrates", NRL Report 7437, 20 July 1972.

2. Private communication to Dr. Homer W. Carhart, NRL August 1973.

3. 3M Company ltr to NAVMAT dated 29 Feb. 72.

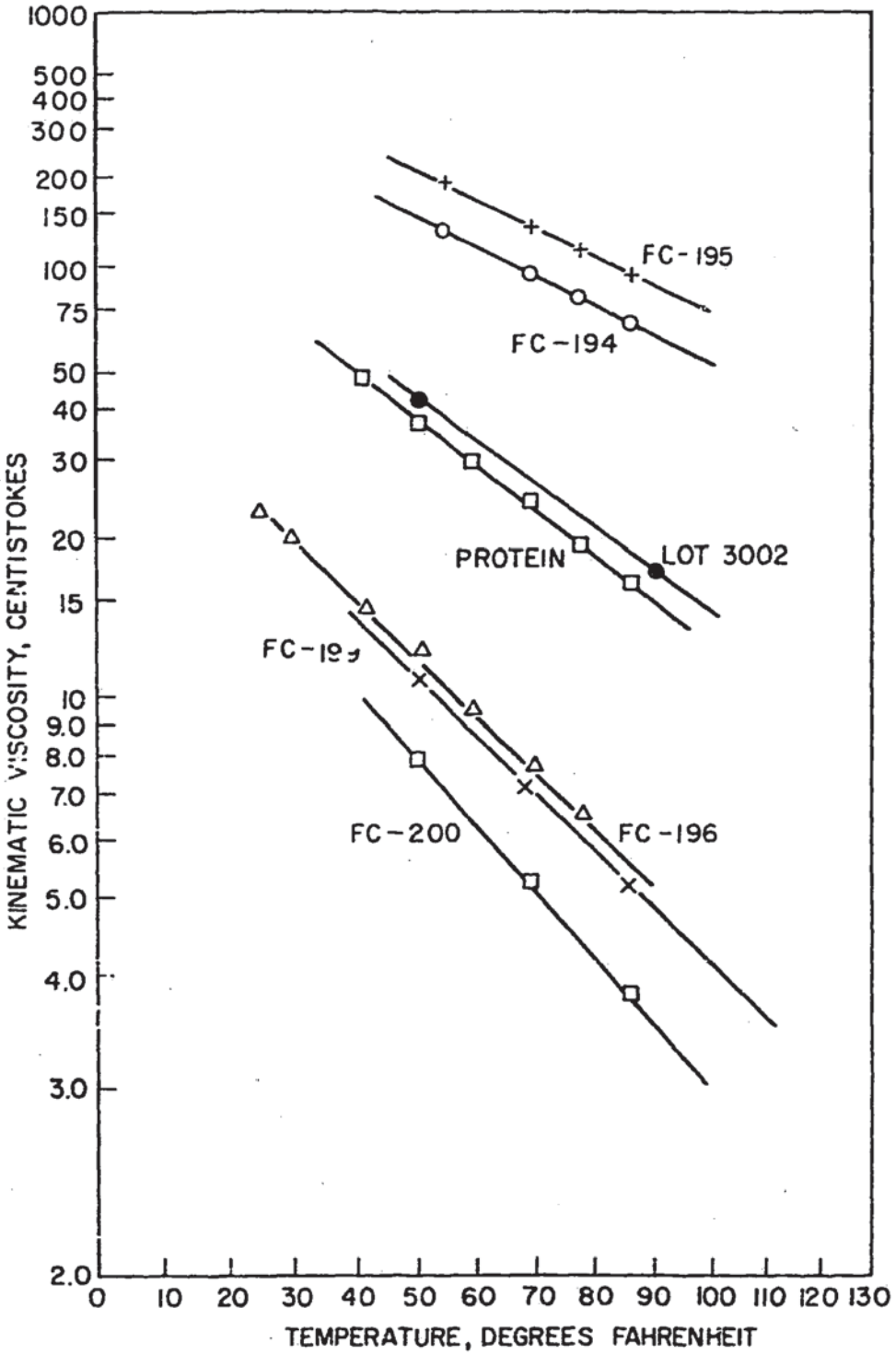

Fig. 1 – Viscosity of AFFF concentrates as a function of temperature.

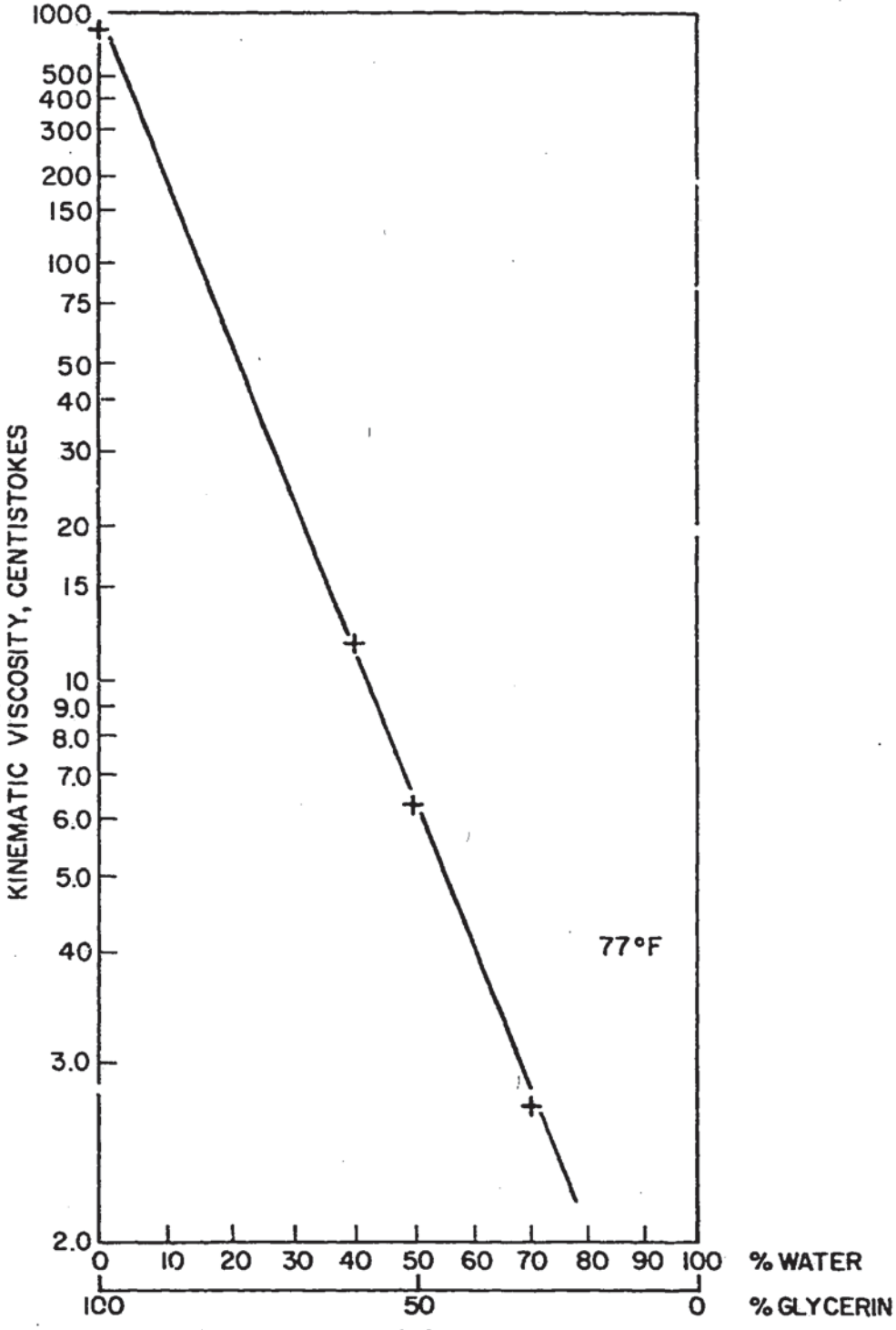

Fig. 2 – Viscosity of glycerin-water mixtures.

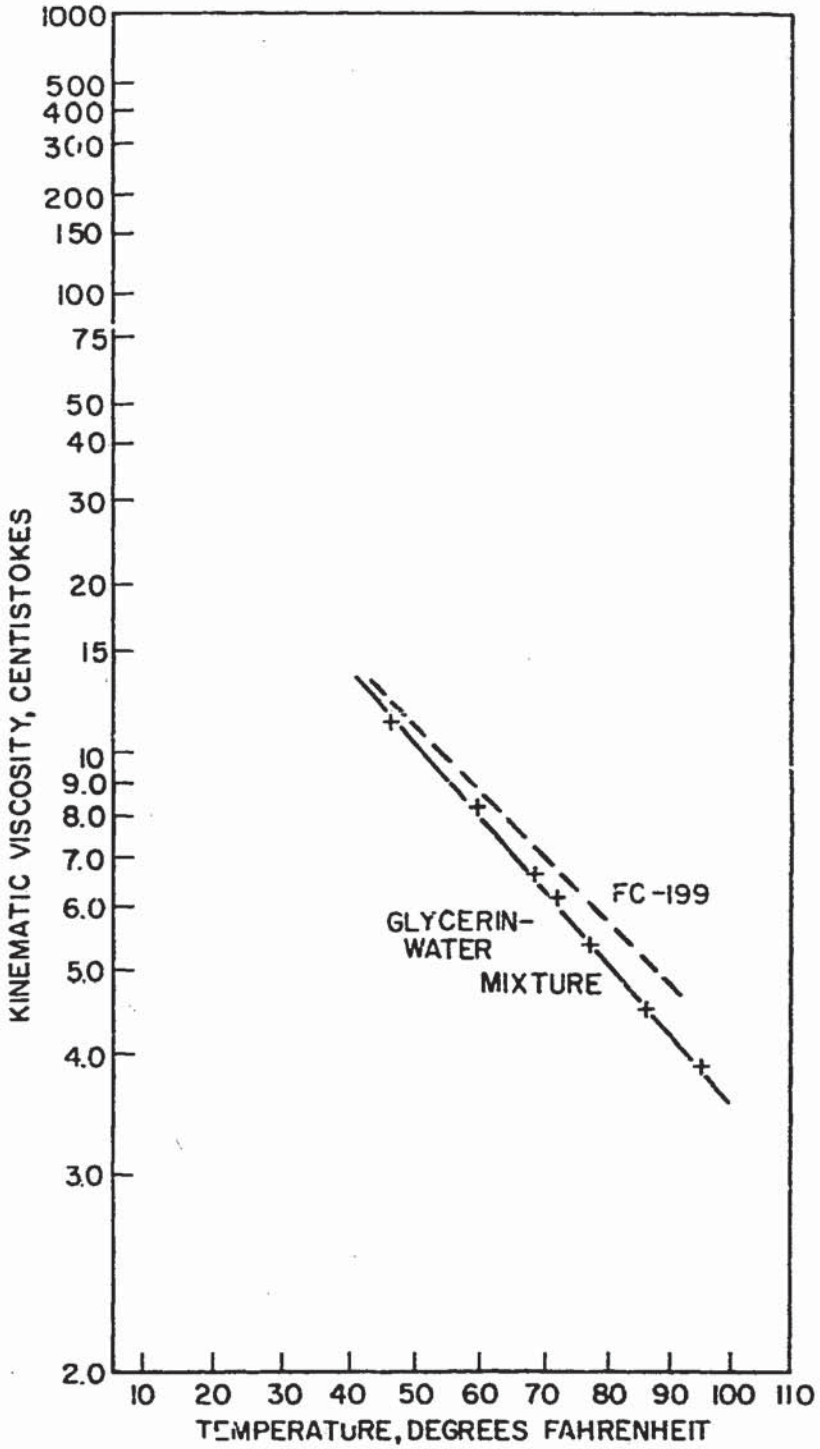

Fig. 3 – Viscosity-temperature relationships of an AFFF concentrate and a glycerin-water mixture.

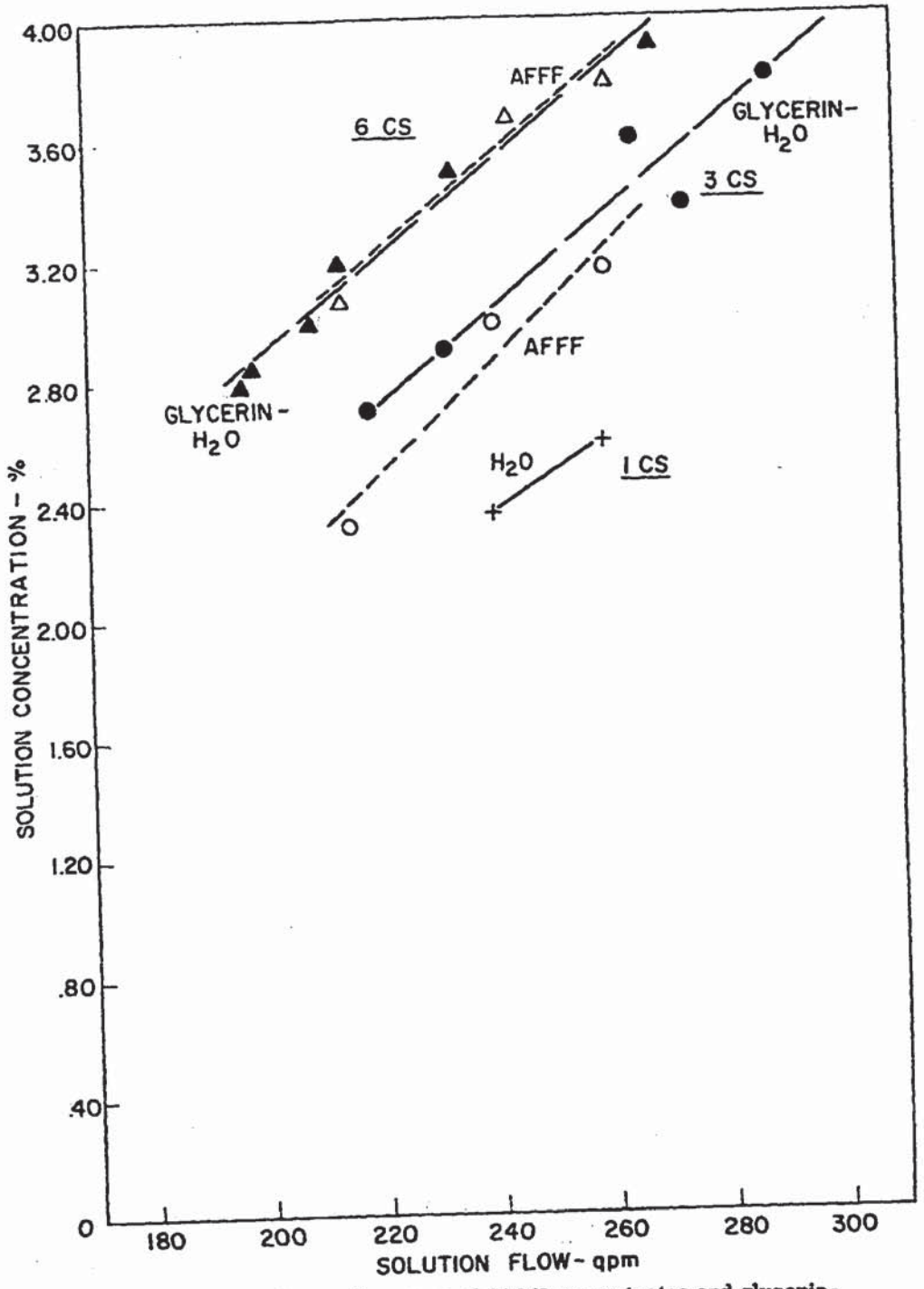

Fig. 4 - Comparative performance of AFFF concentrates and glycerin-water simulated concentrates in an FP-1000 proportioner.

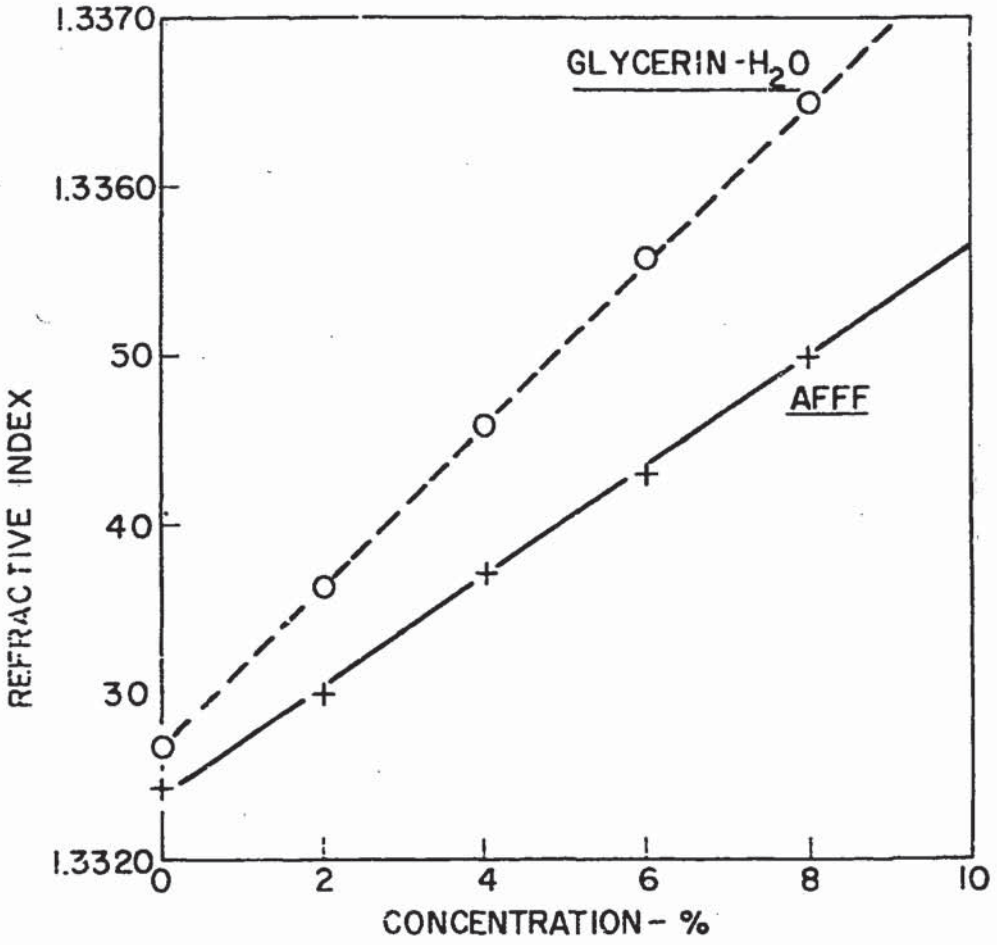

Fig. 5 – Refractive index of solutions prepared from an AFFF concentrate and a simulated concentrate of glycerin and water.

Exhibit 10

28 March 1999

To: 3M

I resign my position as Environmental Specialist effective 6 April 1999. My resignation is prompted by my profound disappointment in 3M's handling of the environmental risks associated with the manufacture and use of perfluorinated sulfonates (PFOS)(CAS# 29081-56-9) and its precursors, such as ethyl FOSE alcohol (CAS #1691-99-2) and methyl FOSE alcohol (CAS #24448-09-7).

Perfluorooctansefulfonate is the most insidious pollutant since PCB. It is probably more damaging than PCB because it does not degrade, whereas PCB does; it is more toxic to wildlife; and its sink in the environment appears to be biota and not soil and sediment, as is the case with PCB.

I have worked within the system to learn more about this chemical and to make the company aware of the dangers associated with its continued use. But I have continually met roadblocks, delays, and indecision. For weeks on end I have received assurances that my samples would be analyzed soon--never to see results. There are always excuses and little is accomplished. I can illustrate with several examples.

- For more than twenty years 3M's ecotoxicologists have urged the company to allow testing to perform an ecological risk assessment on PFOS and similar chemicals. Since I have been assigned to the problem a year ago, the company has continued its hesitancy.
- Over a period of seven months I made frequent requests that ecological risk consultants be hired to help me plan toxicity testing, environmental sampling, chemical fate studies, and ecological risk procedure. I still have not received authorization even to bring people in to interview.
- I requested, very frequently, over a nine-month period, a sample of chemical to send out for fate property and ecotoxicity testing. Finally I was provided with one that apparently the division had had all along.
- I put together a pioneer risk assessment on PFOS that indicated a greater than 100% probability of harm to sea mammals, based on preliminary data on the concentration of PFOS in menhaden fish meal. The 8e committee told me that they would like to reconsider the assessment after we had a validated value for fishmeal. That analysis was given high priority by the committee. After three months the analysis is still not done--not because there were technical problems, but because management did not actually give the analysis high priority.
- 3M submitted a TSCA 8e last May. There is tremendous concern within EPA, the country, and the world about persistent bioaccumulative chemicals such as PFOS. Just before that submission we found PFOS in the blood of eaglets--eaglets still young enough that their only food consisted of fish caught in remote lakes by their parents. This finding indicates a widespread environmental contamination and food chain transfer and probable bioaccumulation and bio-magnification. This is a very significant finding that the 8e reporting rule was created to collect. 3M chose to

report simply that PFOS had been found in the blood of animals, which is true but omits the most significant information.

- One of our customers, Griffin, has data on some of our chemicals. They developed this data for pesticide registration purposes. I started regularly asking for permission to visit Griffin and view the data last May. Their data can help us plan our studies of similar chemicals. It can also indicate if there is an unforeseen risk to certain biota or via certain exposure pathways. It was ten months before I was allowed to visit Griffin, at which time I did not get to see the data. I have to return another time to see it.
- 3M waited too long to tell customers about the widespread dispersal of PFOS in people and the environment. We knew before May of 1998, yet 3M did not start telling customers until January of 1999. I felt guilty about this and told customers I personally knew earlier. Still, it was not as early as it should have been. I kept waiting for 3M to do its duty, as I was continually assured that it would. Some of the customers have done risk assessments on the PFOS precursor they use. They assume there is not a background in the environment and in wildlife. Since there is a background, their risk assessments are inaccurate. Thus they can make inappropriate business decisions and not realize that their use of PFOS precursors contributes to an aggregate risk.
- 3M continues to make and sell these chemicals, though the company knows of an ecological risk assessment I did that indicates there is a better than 100% probability that perfluorooctansulfonate is biomagnifying in the food chain and harming sea mammals. This chemical is more stable than many rocks. And the chemicals the company is considering for replacement are just as stable and biologically available. The risk assessment I performed was simple, and not worst case. If worst case is used, the probability of harm exceeds 100,000%.

- 3M told those of us working on the fluorochemical project not to write down our thoughts or have email discussions on issues because of how our speculations could be viewed in a legal discovery process. This has stymied intellectual development on the issue, and stifled discussion on the serious ethical implications of decisions.

I have worked to the best of my ability within the system to see that the right actions are taken on behalf of the environment. At almost every step, I have been assured that action will be taken—yet I see slow or no results. I am told the company is concerned, but their actions speak to different concerns than mine. I can no longer participate in the process that 3M has established for the management of PFOS and precursors. For me it is unethical to be concerned with markets, legal defensibility and image over environmental safety.

Sincerely,

Rich Purdy